Ambient air quality and the risk of acute myocardial infarction hospitalizations in Lautoka city, Fiji

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Abstract

Background: Air pollution is a known risk factor for heart disease. Results from epidemiological studies from major Western cities suggest that increase in air pollution leads to an increased risk of hospitalisation due to acute myocardial infarction; the increased risk is attributed to the ambient air quality. However, there is limited evidence of the association between air quality and risk of heart disease related hospitalisations in the Pacific. This thesis has addressed the gap in the evidence by conducting a meta-analysis of observational epidemiological studies on the association between air quality and hospitalisation due to acute myocardial infarction or due to significant ischaemia and developed a predictive model based on air quality of Lautoka city and the estimates from the meta analysis. The goal of this thesis is to assess the ambient air quality in Lautoka Central Business District (CBD) and model the risk of hospitalisation due to acute myocardial infarction to the wider population of Lautoka who commute daily into the city centre.

Methods: Ambient air quality was measured for a period of three months from 15th August 2018 to 15th November 2018. These included particulate matter with an aerodynamic diameter less than 10 microns (PM_{10}), particulate matter with an aerodynamic diameter less than 2.5 microns ($PM_{2.5}$), sulphur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO) and ozone (O_3). Air quality data for the Lautoka central business district was analysed and compared with the guideline values set by the World Health Organization. Comparisons were made for the daily mean concentrations as well as mean concentrations during hours of the day over a 24-hour period. A systematic review and meta-analysis of published studies on the association between ambient air quality and risk of hospitalization due to myocardial infarction was conducted which yielded 20 case-cross over studies. On the basis of the ambient air quality data and extrapolation of pooled estimates from the meta-analysis, a predictive model was constructed for those days that would be expected to have high levels of myocardial infarction or acute heart disease related hospitalisations.

Results: Particulate Matter (PM_{10} and $PM_{2.5}$) were the only air pollutants that showed substantial concentrations. The daily mean concentrations observed in the Lautoka CBD for PM_{10} and $PM_{2.5}$ was 64.15μg/m^3 and 9.34 μg/m^3 respectively. The daily (24hr) mean concentrations for PM_{10} exceeded the WHO guideline value of 50 μg/m^3 from Tuesdays to Fridays and the concentrations showed an increasing trend from Mondays to Fridays. Both pollutants exhibited high concentrations during the peak traffic hours (6am-9am in the morning and 4pm to 7pm in the afternoon. Among the 137, 846 AMI hospitalizations the meta-analytic odds in the overall analyses; fixed effects model, OR = 1.04(95%CI = 1.03 – 1.04), random effects model OR = 1.07 (95%CI: 1.05 – 1.10). Both models were significant when analysing exposure to common air pollutants and risk of AMI hospitalization. In the subgroup analysis for short-term exposure to increments of at least 5-10μg/m^3 of $PM_{2.5}$, the effect estimate in the (1) fixed effects model, OR = 1.04(95%CI = 1.03-1.05), (2) random effects model, OR = 1.05(95% CI: 1.03-1.07) respectively. Again, both models were significant when analysing short-term (lag 3hrs -1 day) exposure to $PM_{2.5}$ and risk of AMI hospitalization.

Conclusion: On the basis of the pooled estimates from the meta-analysis and the air quality data, exposure to common air pollutants may increase the risk of AMI hospitalizations among the daily commuters to Lautoka CBD as well as to the wider Lautoka city populace. Importantly, low concentrations of $PM_{2.5}$ was associated with the risk of increased AMI hospitalizations. Furthermore, the risk of AMI hospitalisation is highest on Thursdays and Fridays and 1-24 hrs after exposure to high pollutant levels during peak traffic hours on these days.

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ABBREVIATIONS

PM	-	Particulate matter
WHO	-	World Health Organization
USEPA	-	United States Environment Protection Agency
CVD	-	Cardiovascular disease
CBD	-	Central Business District
IT-1	-	Interim target 1
PICO	-	Participant intervention comparison outcomes
AQMS	-	Air quality monitoring sensor
AQT	-	Air quality transmitter
hPa	-	Hectopascal
SGA	-	Small for gestation age
PM_{10}	-	Particulate matter with and aerodynamic diameter less than 10 (<10) microns
$PM_{2.5}$	-	Particulate matter with an aerodynamic diameter less than 2.5 (<2.5) microns
AMI	-	Acute myocardial infarction
ACS	-	Acute coronary syndrome
ppm	-	Parts per million
ppb	-	Parts per billion
STEMI	-	ST elevation myocardial infarction
TEOM	-	Tapered element oscillating microbalance
GLMM	-	Generalized mixed linear models
UFP	-	Ultra fine particles
SIDS	-	Small Island Developing States

CHAPTER 1

INTRODUCTION

1.1 Summary

This chapter has provided the platform on the perspectives that warrants the commencement of this study. An introduction into the thesis topic is presented as well. Global and local perspectives on ambient air quality and cardiovascular health has been briefly explained. The aims and objectives that will be the focus of this research has been presented. The rationale for the research based on evidence from epidemiological studies and the lack of such studies has been highlighted validating the purpose of this research and a brief description of the chapters in this thesis.

1.2. Introduction

In 2016, ambient air pollution was responsible for an estimated worldwide deaths of 4.2 million people of which 16% of the deaths are from lung cancer, 25% from chronic obstructive pulmonary, 17% from ischemic heart disease and stroke and 26% from respiratory infection (WHO, 2019). Air pollution in most developing countries originate from sources such as combustion processes from motor vehicles, open burning of wastes and emissions from industrial processes. Most of these emissions contain a heterogeneous mixture of pollutants known as criteria pollutants or common pollutants and includes particulate matter (PM), ground-level ozone, carbon monoxide, lead, sulphur dioxide and nitrogen dioxide(USEPA, 2018). These pollutants are of major health concern because of the various cumulative effect it has on the respiratory and cardiovascular health. Principal among these pollutants are particulate matter with an aerodynamic of <10microns (PM_{10}) and particulate matter with an aerodynamic diameter of <2.5microns ($PM_{2.5}$) which has been reported to cause acute cardiovascular and respiratory morbidity and mortality (Pope & Dockery, 2006).

Globally, heart diseases are the leading cause of deaths with more people dying from it than from any other cause. The World Health Organization estimates that in 2016, 17.9 million people died from heart diseases, representing 31% of all global deaths. Eighty five percent (85%) of these deaths are due to heart attack and stroke and three quarters of CVD deaths occur in low- and middle-income countries (WHO, 2018). Moreover, of the 17 million premature deaths for people under the age of 70 due to non-communicable diseases, 82% were in low- and middle-income countries and 37% of these deaths were caused by cardiovascular diseases.

Air pollution is increasingly recognized as a risk factor for cardiovascular disease. Recent epidemiological studies and evidence have consistently shown the increased risk of cardiovascular disease with exposure to air pollutants. In 2004, the American Heart Association (AHA) Scientific Statement concluded that exposure to particulate pollution contributes to cardiovascular illnesses and deaths, especially for myocardial infarction, stroke, cardiac arrhythmia and heart failure (Brook et al., 2004).

Ambient air quality in all the urban areas in Fiji are not monitored, hence, it is difficult to quantify the level of pollutants and the associated risks. Although it is a known risk factor to the triggering of cardiovascular events in developed countries, not often is it perceived to that effect in Fiji because of potentially insufficient epidemiological evidence. Heart diseases is the major cause of premature deaths in Fiji and the causes are often associated with the traditional

risk factors such as high blood pressure, high cholesterol level, unhealthy diet, physical inactivity and excessive consumption of alcohol.

The outcome of these thesis aims to build the body of knowledge in Fiji pertaining to the risk of cardiovascular events after a short-term exposure to common air pollutants. In essence, this study will examine the ambient air quality in the Lautoka Central Business District (CBD) and the risk of acute myocardial infarction hospitalizations and emergency department visits.

1.3 Ambient air quality in Fiji

The Republic of Fiji is one of the developing nations nestled in the south pacific with an estimated population of 884, 167 people. Fifty seven per cent (57%) of these total population live in the urban areas and 43% live in the rural areas (FBoS, 2018). There is no monitoring of ambient air quality in any city or town in Fiji that would provide an insight of the actual level of pollutants present in its immediate surroundings. However, industrial and commercial occupational activities that may release significant emissions are required to carry out their own air quality monitoring and provide this monitoring data to the Department of Environment in Fiji for verification and recording purposes only.

In 2013, the Department of Environment in Fiji (DoE) collected information on potential air pollution sources through qualitative means. Qualitative data was obtained from public complaints, visual observations and local studies. From this data, the Department of Environment of Fiji established that the main sources of pollution in terms of the volume emitted are from vehicle emissions, burning of dumpsites, burning of household waste, backyard burning, agricultural burning, emissions from incinerators, industrial emissions, cooking in open stoves and dust from unpaved roads.

To date, the only study on air quality in Fiji that has been published was carried out in 2015 by Isley and others. Their study focused on measuring ultrafine particle concentrations (PNCs) in order to demonstrate combustion emissions. The study by (Isley, Nelson, & Taylor, 2016) on the air quality in Suva found that $PM_{2.5}$ concentrations complied with the WHO annual average of 10 $\mu g/m^{3.}$ However, black carbon (BC) levels in the city of Suva were much the same in cities in more industrialized countries like New Zealand, Australia, England and Ireland. Isley and Taylor concluded that the increased vehicular traffic and increasing industrial activities immensely contribute to the particulate pollution in the city. Added to this is the widespread burning of wastes and the emissions from low grade second-hand vehicles and older diesel vehicles.

1.4 Cardiovascular Health in Fiji

Cardiovascular diseases accounts for 17.7 million deaths each year which is estimated to be 31% of all deaths worldwide. Developing nations like Fiji are experiencing a change in disease patterns with a significant decline in infectious diseases and a corresponding increase in non-communicable disease such as diabetes, cardiovascular disease and cancer. Non-communicable diseases (NCDs) accounts for an estimated 84% of all deaths of which 34 % are attributed to cardiovascular diseases and 22% from diabetes in Fiji (WHO, 2018). NCDs are the leading cause of deaths in Fiji and despite the fact that diabetes is not the major cause of death, it is still a major concern. Cardiovascular diseases that are of concern in Fiji includes ischemic heart disease, stroke, hypertensive heart disease, cardiomyopathy and myocarditis

and rheumatic heart disease. Of these cardiovascular diseases the most common in Fiji is ischemic heart disease or coronary heart disease which includes heart conditions such as angina and myocardial infarction (IHME, 2018).

In 2018, the Ministry of Health and Medical Services in Fiji and the World Health Organization established that cardiovascular disease is the leading cause of deaths in Fiji. There are almost twice as many cardiovascular related deaths in Fiji as those from diabetes (PACNEWS, 2018). The major risk factors for cardiovascular diseases in Fiji are smoking, having an unhealthy diet, lack of physical activity and misusing of alcohol. In addition, a person becomes more at risk of cardiovascular disease if they have raised blood pressure, increased blood sugar and are overweight or obese.

The Ministry of Health and Medical Services and WHO (development partner), are leading the fight against non-communicable diseases. Early identification of the individuals who are at risk of developing these diseases is utmost important and is one of the main reason that all health facilities in Fiji are offering free screening services for NCDs and identifying underlying risk factors. NCD screening is also provided in workplaces and communities so that risks are identified for individuals and advice on mitigating measures are given to make changes needed to prevent these life threatening diseases.

1.5 Description of Lautoka City

The city of Lautoka is the second largest of the two cities in Fiji, and is nestled on the western part of Fiji's main island, Viti Levu (refer to Fig 1). Lautoka is located 24 kilometers north of the town of Nadi where Fiji's International airport is located. The city lies in the heart of Fiji's sugar cane growing region and is affectionately known as the 'Sugar City' of Fiji. It covers an area of 16 square kilometers and is home to Fiji's largest sugar mill. The city has an estimated population of 52,500 people from various social and ethnic backgrounds. The ethnic distribution of the population consists of indigenous Fijians which accounts for 43% of the population, Fijians of Indian descent accounts for 45% and people from other ethnic background makeup the remaining 7% of the population.

Figure 1: Illustrates the four divisions in Fiji and Lautoka City on the Western side of Viti Levu Source: Google Maps

As the second largest city, Lautoka harbour's most industrial and commercial enterprises in the Western part of Fiji's main land. This include timber mills, breweries, oil/ghee refining factory, aerated water and juice factory, concrete industries, soap factory, engineering and steel workshops, flour mill, bakeries, etc.

The weather in Lautoka is similar to that of in Nadi as both are within close proximity. The rainy season is usually between the months of December to April and precipitation is quite low as compared to the capital of Fiji, Suva which is located in the central division. The average rainfall in a year is estimated at 2,050mm or 80.5 inches per year and during the dry season (May to October), rainfall can be as low as 100mm or 4 inches per month. The temperature on western part of Viti Levu is a little higher than other locations in Fiji with a mean annual temperature of 28^0C. The amount of sunshine too is higher in Lautoka which enjoys 7-8hrs of sunlight per day as compared to Suva which has an average of 5-6 hours of sunlight.

1.6 Rationale for research

Globally, a lot of research has been conducted and focused on urban population in developed countries and where issues relating to air pollution is readily apparent. Ambient air quality in developing countries has always been of concern because of the continuous increase in urban development, increasing population and the continued reliance on fossil fuels as the main source of energy. Most of the populace in developing counties live in the rural areas whereby households still rely on unprocessed biomass fuels in the form of wood and crop residues. The gaseous pollutants released from the combustion of these energy sources have varying and significant effects on human health and one that is of importance to this study is the effect it has on cardiovascular health. As a developing country, the most commonly known or traditional risk factors to cardiovascular diseases in Fiji are being overweight or obese, smoking, high blood pressure and high cholesterol levels, being physically inactive, having an unhealthy diet, diabetes and prediabetes and having a family history of early heart disease.

My area of specialization in Fiji is Environmental Health, hence a systematic investigation of the association between an environmental variable (pollutants in air) and a health effect (acute myocardial infarction) will provide the nous amongst health researchers in Fiji on the influence of environmental factors on cardiovascular health. Air pollution is one of the largest risk factors that lead to a range of diseases. Heart disease is a common cause of death and disability worldwide. In Fiji alone, 34% of annual deaths is from cardiovascular diseases (WHO, 2018). Being from Fiji and an ardent advocate on impacts of environmental exposures on human health, this research will provide the impetus in developing explanatory models of the linkage between air quality and risk of heart disease among the populace in an urban area.

This research is unique in the sense that it will be one of the first studies to quantitatively examine the effects of exposure to air pollutants in an urban area in Fiji and risk of hospitalization due to acute myocardial infarction. The study will put into perspective the growing epidemiological evidence on pollutant levels and risk of hospitalizations from acute myocardial infarction for the populace that commute daily to the city of Lautoka. Investigating the outcome of this study in order to develop policies and plans for ambient air quality is not within the scope of this thesis. This research is purely to examine the ambient air quality and risk of acute myocardial infarction hospital admissions in Lautoka.

1.7 Research aims

The general aim of this research is to determine if exposure to increased levels of criteria pollutants (particulate matter with an aerodynamic diameter of <2.5microns and <10 microns, carbon monoxide, Sulphur dioxide, nitrogen dioxide and ground-level ozone) will lead to increased rate of emergency room hospitalizations due to heart disease (also referred to as acute myocardial infarction in this thesis). To achieve this, three principal objectives were developed to be the focus of this research and are as follows:

- Collection and determining of the ambient air quality in the Lautoka Central Business District for a period of three months and its potential health implications
- Conducting a meta-analysis of published studies on the association between ambient air quality and risk of hospitalization due to myocardial infarction.

Lastly, extrapolations on the risk of AMI hospitalizations will be made on the basis of the meta-analysis and air quality data collected.

1.8 Thesis outline

The layout and content of this thesis is generally constructed to satisfy the aims of this research. Chapter One has introduced the research topic and the rationale for conducting the study. The general aims of the research and the study site has also been introduced thoroughly in this chapter. In addition, a brief on the ambient air quality in Fiji and the outlook of cardiovascular health was also presented. Chapter two provides the literature review of what is known about the research topic. It examines the history of air pollution and the associated cardiovascular effects. In particular the health outcome of interest, acute myocardial infarction. Also provided here are the air pollution issues in Fiji and the drawbacks that the country has in terms of measuring and addressing air quality. Chapter 3 introduces the study design adopted for this research and the methods selected to obtain the necessary quantitative measurements for this research. In particular, this part emphasizes the analysis methods used to provide precise pooled estimates in the meta-analysis. Chapter 4 provides the qualitative and quantitative output of all the approaches and procedures used in chapter 3 and sets the scene for chapter 5. In this part, the exposure and outcome variables are combined and analyzed to determine the relationship between them In particular it summarizes the air quality data and provides a descriptive analysis. The analysis used to obtain the summary effects is also outlined in this chapter. Chapter 5 elaborates on the findings observed in chapter 4. It explains how the pollutants levels are in the Lautoka CBD and its health implications. Importantly it synthesizes the air quality data with the summary effects obtained from the meta-analysis and extrapolate these findings to the daily commuters to the Lautoka city and risk of hospitalizations. Chapter 6 provides some inferences of how the findings from these study will impact the wider population of Lautoka city. Some limitations observed during this study is also highlighted as well as possible opportunities that can emanate as a result of the findings from this study.

CHAPTER 2

LITERATURE REVIEW

2.1 Summary

This chapter has chronologically highlighted the evolution of air pollution over the past centuries to the current one. An overview of how the sources of air pollution has changed over the years is also discussed with particular reference to those used in transport systems and industries. An update on recent air pollution status from a global perspective is briefly described. In addition, the characteristics of common air pollutants or criteria pollutants that is of global concern is also deliberated herein with their potential sources. The last segment of this chapter deliberates on the potential health effects of ambient air pollution with specific reference to how it impacts the cardiovascular health

2.2 Introduction

The objective of this chapter was to conduct an assessment on the body of knowledge relating to air pollution and cardiovascular health. The chapter commences with a brief history on the evolution of air pollution before the industrial revolution to the current air pollution problems and challenges. In addition to this is that it elaborates on how the advancement of technology has in a way mitigated the effects of air pollution and emission of pollutants in developed countries but not so much in developing countries and small islands developing states (SIDS). Also presented in this chapter are the cardiovascular effects of air pollution and how it has impacted the cardiovascular health of the population in certain countries described herein.

2.3 History of Air Pollution

2.3.1 Before the Industrial Revolution

Air pollution can be traced back to the tribes in early history whereby they lived nomadic lives to avoid the stench of decaying animals as well as foul odour from vegetable and human waste. These early tribesmen also learned to use fire and this new knowledge was quite important in their daily lives. The usage of fire would fill the inside of their living homes with the products of incomplete combustion and examples of this can still be seen in some primitive parts of the world (Vallero, 2008). Later on, the invention of chimneys removed the combustion materials and cooking odours from the livable spaces.

In the bronze and iron ages, industries associated in the generation of air pollution such as dust and fumes were from metallurgy, ceramics and preservation of animal products. These industries were responsible for the production of clay and bricks before 4000BC. The production and use of iron came into effect around 1000BC. During this period people relied on the use of charcoal as a source of fuel rather than coal or coke (Boubel et. al., 1994). The burning of wood in fireplaces inside homes created emissions that were smoky and in AD 61 Roman philosopher Seneca said that,

"As soon as I had gotten out of the heavy air of Rome and from the stink of the smoky chimneys thereof, which, being stirred, poured forth whatever pestilential vapours and soot they had enclosed in them, I felt an altercation of my disposition"

Later on in 1157, the wife of King Henry II of England, Eleanor of Aquitaine, decide to move away from Tutbury Castle because she cannot endure the burning of wood. One hundred and sixty years later the burning of coal was prohibited in London and in 1306 Edward I issued a Royal proclamation authorizing the use of sea coal in furnaces. By 1661, air pollution in London was a huge problem and it prompted John Evelyn to advise the parliament and King Charles II on the quality of air in London with possible remedies to control air pollution. These propose remedies are still relevant now in the 21st century (P.J.B., 1956).

2.3.2 The Industrial Revolution

The Western civilization embraced the industrial revolution because it brought prosperity, social changes and altered the direction of their history. Its' disadvantage was the over reliance of industries on coal as an energy source and the severe air pollution that it caused (Air pollution and health 1999). Harnessing of steam to move machinery and pump water began in the early years of the 18th century and culminated in 1784 with Watt's reciprocating engine that was later replaced with the steam turbine in the twentieth century (*Fundamentals of Air Pollution*, 1973).

A consequence of this era was a lot of pollution from the smoke and ash from the burning of coal in the boiler furnaces of stationary power plants, locomotives, home heating fireplaces and furnaces. In 1819, Great Britain took the initial steps to address the problem brought about by air pollution and in 1856 saw the creation of laws specifically for London. These laws were introduced to reduce the imminent threat of air pollution and deemed the emission of smoke as a public health nuisance (Beaver, 1955). In 1880, the United States of America developed municipal ordinances and regulations targeting the emission of black smoke and ash from industrial, marine and locomotive sources. Despite the introduction of new laws to curb the emission of smoke and ash in major industrialized countries, air pollution was at its worst towards the end of the 19th century. The principal engineering technological advancement in the control of air pollution were the stoker for mechanical firing of coal, scrubber for removing acid gases from effluent gas steams, cyclone and bag house dust collectors, and the introduction of physical and chemical principles into process design (Boubel et. al., 1994)

2.3.3 The 20th Century

In the early 1900s, there was still a reliance on the use of coal but technological advances such as the replacement of the steam engine with the electric motor was a major breakthrough. Towards the end of the 1st quarter of this century, the use of coal was being replaced by pulverized coal, oil and gas. But each of these new energy sources produce their own characteristic emissions to the atmosphere. In this period there was a decrease in ash emissions as oil had replaced the use of coal resulting in the significant increase in automobile production. The period 1925 to 1950 saw the emergence of the present day pollution problems such as the Meuse Valley Smog in 1930 whereby a combination of air pollution and climatic conditions killed sixty people (Firket, 1936). In 1948, an air inversion (an air inversion is an incident in which air stops circulating and cool is trapped close to the ground containing a combination of

toxic gases and early morning mists that has the potential to be harmful to human health) episode similar to that shown in Figure 2 known as the Donora Smog in Donora, Pennsylvania killed almost 40 people and left nearly half of the town's 14,000 residents with severe respiratory and cardiovascular problems (USEPA, 2017). The city of Los Angeles also experienced the effects of smog in the 1940s as a result in the influx of cars and industries combined with a geography that traps fumes.

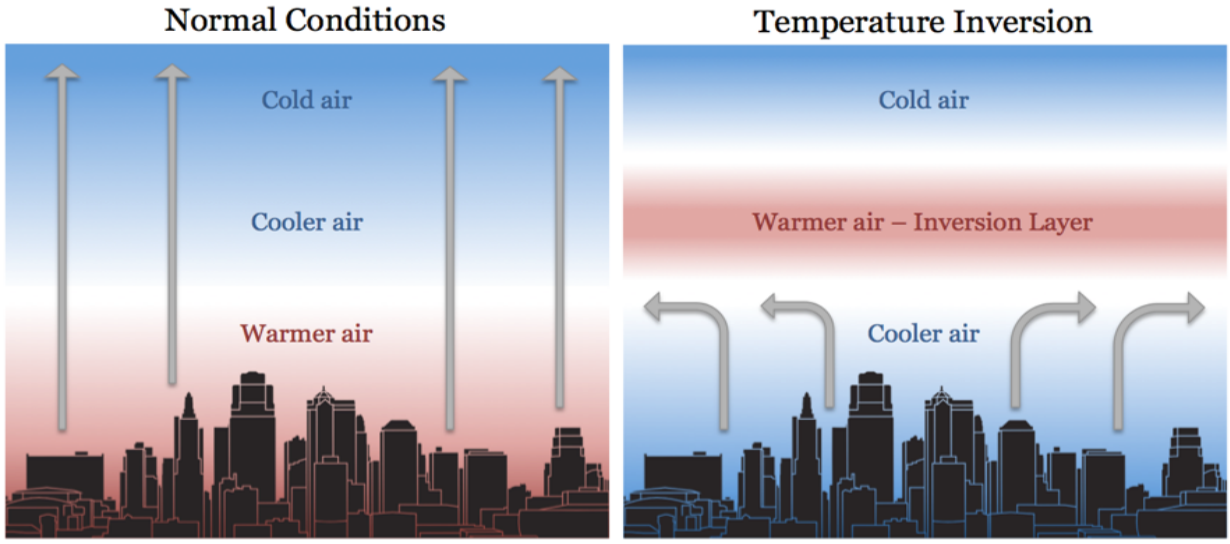

Figure 2: Picture on the left shows air flow in normal conditions and on the right is the air flow during a temperature inversion Source: Cotton NC State University; permissions to use photos and graphics applied for

Towards the end of the first half of this century, major technological advancement and changes were made in major industrial countries like the United States and Great Britain. A good example was in Pittsburgh, Pennsylvania whereby the use of coal and oil as home heating fuel was substituted with the use of natural gas. In addition to this was the displacement of steam locomotives and replaced with diesel locomotives. Despite the growing problem of air pollution during the period 1925-1950, no country in the world adopted any pollution laws to try and mitigate harmful emissions. It was the state of California that enacted the first state air pollution law in the United States.

The period 1950-1980 was marked with a major pollution disaster in Great Britain which was later known as the 'Great London Smog'. During this period coal was the main source for generating power and heating homes in London. It was on the 5th of December 1952 that a high pressure weather system stalled over southern London and caused a temperature inversion. For five days, a heavy fog combined with smoke from the fumes of vehicle exhaust and power plants created a public health disaster which resulted in an estimated of 4,000 premature deaths (Klein, 2012). The impact of the London Smog provided the impetus for the regulating of emissions with the enacting of the Clean Air Act of 1956. The new law required the markets to provide cleaner fuels for home owners, phasing out of coal in urban areas, power stations closed to residential areas were closed and height of chimneys were regulated. As a result of high air pollution levels, almost all European countries including Australia, New Zealand and Japan have created legislation to control air pollution. In the United States, federal laws were

introduced in 1955 to support research in air pollution, training and technical assistance (Fundamentals of Air pollution 1984). Later in 1970, the United States Environment Protection Agency (USEPA) came into existence with the federal mandate of protecting the environment and human health. The second half of the twentieth century also marked the increase in air pollution from automobiles, pollution from flue gases and emissions from combustion (Vallero 2008*).* In the 1970s, the buildup of greenhouse gases was a major concern as it was found to be contributing to the depletion of the stratosphere ozone layer. Also, the discovery of the Antarctic 'ozone hole' in 1985 and the emergence of uncontrolled changes in climate in 1990s saw the emergence of the major problem brought about by greenhouse gases such as chlorofluorocarbon (CFCs)(Elsom, 1992). Towards the end of this period, air quality monitoring systems and modelling of atmospheric processes came of age and highlighted the global concern of air pollution. Subsequently, the scientific community and the general public has taken a greater urgency on global air pollution since the early years of the 21st century.

Table 1: Ranges of annual average concentrations of PM10, NO2, SO2 and 1 hr. average maximum of ozone for different regions. Source: WHO

Region	Annual average concentration			Ozone (1hr max concentrations)
	PM_{10}	NO_2	SO_2	
Africa	40-150	35-65	10-100	120-300
Asia	35-220	20-75	6-65	100-250
Australia/New Zealand	28-127	11-28	3-17	120-310
Canada/ United States	20-60	35-70	9-35	150-380
Europe	20-70	18-57	8-36	150-350
Latin America	30-129	30-82	40-70	200-600

2.4 Recent Ambient Air Pollution

Air pollution in the industrialized and developing world has changed drastically from the last century as a consequence of the rapid global population growth (Fenger, 2009). Economic development, energy consumption, urbanization, transportation and motorization are other driving forces of air pollution in urban cities (Chen & Kan, 2008). Nevertheless, the urban environment in most industrialized countries has improved from pollution caused by power and heat generation (Fenger, 2009). In the 2005 Air Quality Guidelines Global update, the World Health Organization summarized that the annual average concentrations of PM_{10} in European and North American cities were generally lower than 50$\mu g/m^3$ as shown in Table 1. Asia, Africa and Latin America had the highest levels of PM_{10} compared to other regions and SO_2 has substantially declined in the United States of America and Europe. Decline of SO2 levels in Bangkok, Jakarta and New Delhi is attributed to the use of fuel with low sulphur content. Latin America and Africa also recorder moderate reduction in SO2 levels (Chen & Kan, 2008).

Table 1: Ranges of annual mean levels of common air pollutants for different regions. All measurements are in µg/m^3 Source: WHO

Region	Annual average concentration			Ozone (1hr max concentrations)
	PM_{10}	NO_2	SO_2	
Africa	40-150	35-65	10-100	120-300

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Europe	20-70	18-57	8-36	150-350
Latin America	30-129	30-82	40-70	200-600

In contrast, (Chen & Kan, 2008) suggests that countries in transition have shown that traffic related air pollutants such as NO_2 and SO_2 tend to increase due to increasing number of motor vehicles. Mega cities such as Beijing, Tokyo, Osaka, New York, Los Angeles and Sao Paulo have recorded NO_2 levels that exceed the WHO criteria of $40\mu g/m^3$.

The recent report by WHO on the State of Global Air/2018 estimates that 95% of the world's population reside in areas with unhealthy air and 58% live in areas where the $PM_{2.5}$ concentrations were above the annual WHO interim target 1 (IT-1) of $35\mu g/m^3$. Moreover, 69% of the world's population reside in locations exceeding the interim target (IT-2) of 25 ug/m^3. Countries in North Africa recorded the highest concentrations of population-weighted annual average $PM_{2.5}$ (Niger at $204\mu g/m^3$, Egypt at 126 $\mu g/m^3$), West Africa (Cameroon at 140 $\mu g/m^3$, Nigeria at 122 $\mu g/m^3$) and in the Middle East $PM_{2.5}$ concentrations in Arabia was at 188 $\mu g/m^3$ and Qatar at 148 $\mu g/m^3$. The report has visualized the global population-weighted $PM_{2.5}$ concentrations in Figure 3 below (Health_Effects_Institute, 2018). The sources of these extreme $PM_{2.5}$ concentrations in these regions are mostly due to windblown mineral dust whereas for countries like Niger, Nigeria and Cameron the contribution to the outdoor pollution can also be attributed to the burning of fossil fuels in the home, open burning of agricultural land or forests.

Moreover, the State of Global Air Report/2018 report indicates that the lowest annual average population-weighted $PM_{2.5}$ ($\leq 8\mu g/m^3$)were recorded in Finland, Australia, New Zealand, Canada, Brunei, Sweden, Greenland and several Caribbean and Pacific Island countries.

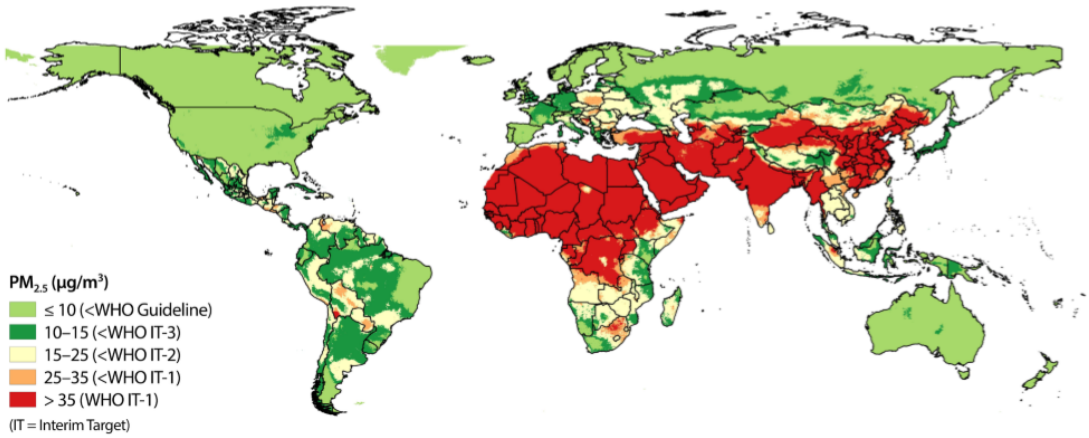


Figure 3: Comparison of the global annual average PM2.5 concentrations in 2017 with WHO Air Quality guidelines Source: Health effects Institute 2018

Furthermore, the report on the State of Global Air/2018 suggests that global weighted $PM_{2.5}$ concentrations has increased by 18% from $43.2\mu g/m^3$ in 2010 to $51.1\mu g/m^3$ in 2016. The rapid increase in the $PM_{2.5}$ concentrations was significant from the year 2010 onwards as illustrated in Figure 4. These increases reflects the changes in air pollution levels in some of

the most populous countries in the world such as China, Bangladesh, India and Pakistan. Even though China had high $PM_{2.5}$ concentrations due to their scale of spurring economic development before 2010, exposures seems to have taken a slight decline. Bangladesh, Pakistan and India currently represents the highest sustained $PM_{2.5}$ levels among the countries represented in the figure 4

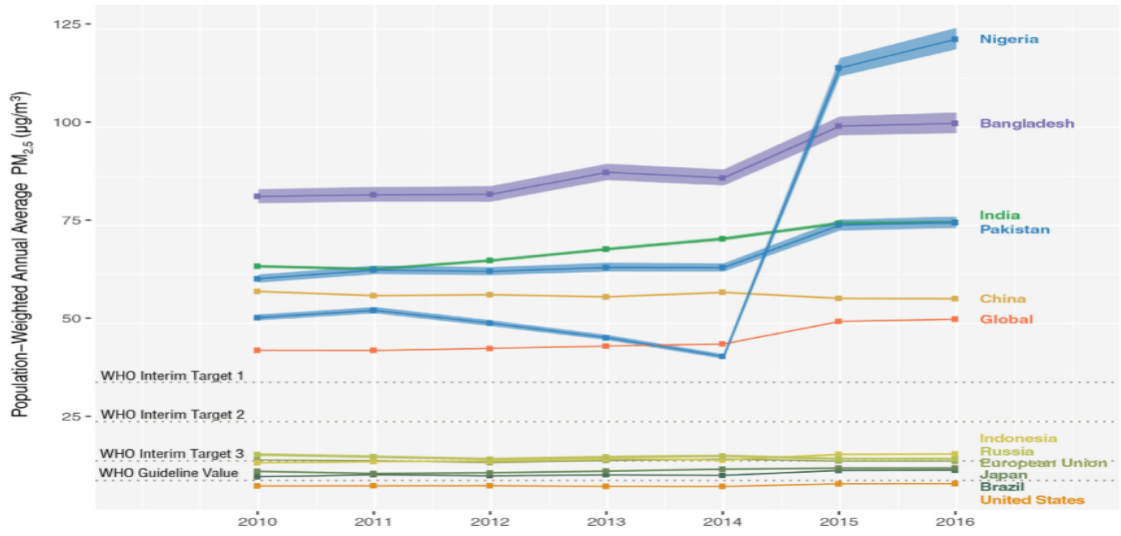


Figure 4: Annual mean population weighted PM2.5 concentrations in the 10 most populous countries including the European Union, 2010-2016 Source: State of Global AIr-2018

The State of Global Air 2017 report adds that the ozone levels have increased by 7% globally from 1990 to 2015. This is due to a combination of factors and principal among them is the increase in emissions of ozone precursors such as NO_2 combined with the warmer temperatures in developing countries like China, Pakistan, Brazil and Bangladesh. The United States and the European Union have noted a decrease in ozone and $PM_{2.5}$ concentration levels by 5% and 2% respectively. The decrease in concentration levels in the USA and EU is due to the air quality management programs that has been in place in these countries since 1990.

2.5 Air Pollution in Developing Countries

Developing countries face particularly difficult choices in balancing efforts to protect the environment with efforts to spur economic growth (Alberini et al., 1997). As a consequence, rapid urbanization and increased migration to urban areas often lead to an influx in traffic and industrial activities. With the increase in traffic and industrial activities, emission of gaseous pollutants are often uncontrolled and contribute significantly to ambient air pollution in the urban areas(Mannucci & Franchini, 2017). Because of economic and other societal reasons, developing nations like Fiji often take a passive role in regulating ambient air quality in its urban areas.

The World Health Organization estimates that in 2016, 4.2 million premature deaths was caused by ambient air pollution in both cities and rural areas. From these deaths, 91 percent occurred in low-and middle-income countries and the greatest burden was in the WHO South-East Asia and Western Pacific regions (WHO, 2018). Modernization has seen a shift from the use of biomass fuel to petroleum products and electricity in developed countries. However, households continue to use biomass fuels and poverty is seen as one of the main barriers to the adoption and use of cleaner fuel sources (Gordon et al., 2014).

Indoor air pollution is a major public health threat and about 50% of the people in developing countries rely on coal and biomass in the form of wood, dung and crop residues for domestic energy. The slow rate of development in most of these developing countries suggests that biomass fuels will continue to be used by the poor in years to come (Nigel Bruce, 2000). Majority of households in developing countries still use earth ovens and stoves whereby incomplete combustion results in substantial emission of pollutants. These cooking practices are inefficient, and use fuels and technologies that produce high levels of household air pollution with a range of health-damaging pollutants, including small soot particles that has the ability to penetrate deep into the lungs. In poor ventilated houses, indoor smoke can be 100 times higher than the acceptable levels for fine particles. Exposure is particularly high among women and young children, who spend most of their time near the domestic hearth (WHO, 2018). In these countries, indoor concentration of particles usually exceed the guideline levels by a large margin (Smith et. al., 1994). These high concentrations of particulate matter and carbon monoxide have shown to be associated with health effects that may occur among the children such as reduced lung function and exacerbation of chronic obstructive pulmonary diseases. This is in stark contrast to developed nations whereby the use of gas stoves has evolved into adopting stoves with cleaner energy sources like electricity

2.6 Characteristics of common Air Pollutants

The air we breathe can contain a variety of pollutants emitted into the atmosphere outdoors (also called ambient air) as well as into the air indoors. Air pollution can contain a mixture of solid particles, liquid droplets and gases from a variety of sources such as industry, motor vehicles, heating appliances, and tobacco smoke (NSW-Health, 2013). The World Health Organization has established that the pollutants with the strongest evidence of health effects are particulate matter are particulate matter ($PM_{2.5}$ & PM_{10}), ground-level ozone (O_3), nitrogen dioxide (NO_2) and sulphur dioxide (SO_2) and that adverse health consequences can occur as a consequence of short/long-term exposure to them(WHO, 2018). Moreover, WHO have established guidelines (refer to Table 2) for these pollutants in order to offer guidance in reducing its health impact and these guidelines are applicable across all WHO regions. Due to their occurrence in the atmosphere, these pollutants are classified as indicator pollutants for fuel combustion and traffic related air-pollution (Chen & Kan, 2008). In the United States of America, the USEPA has categorized these four pollutants and two others, namely carbon monoxide (CO) and lead (Pb) as criteria pollutants. This is because these pollutants are commonly found all throughout the United States of America and has the tendency to cause harm to human health and the environment.

Table 2: Guideline levels of the pollutants that were measured in this study Source: WHO

Guideline levels for each pollutant

$PM_{2.5}$	1 year	10 (µg/m3)
	24 hour mean	25 (µg/m3)
PM_{10}	1 year	20 (µg/m3)
	24 hour mean	50 (µg/m3)
Ozone, O_3	8hour, daily maximum	100 (µg/m3)
Nitrogen dioxide, NO_2	1yr	40 (µg/m3)
	1 hr	200 (µg/m3)
Sulphur dioxide, SO_2	24 hour	20 (µg/m3)
	10min	500 (µg/m3)

To satisfy the aims of this study, these pollutants were measured during the 3 months period in Fiji. Understanding their occurrence and sources (as outlined below) is imperative in addressing air pollution in Fiji.

2.6.1 Particulate Matter (PM10 & PM2.5)

Often called particulates, constitutes a major class of pollutants. Particulates vary in shapes and sizes with a wide range of physical and chemical properties and can be either liquid droplets or dry dusts, (C. David Cooper, 2011). Primary particles are directly released into the atmosphere by wind, combustion processes, or human activities. On the other hand, secondary particles are those that are formed in the atmosphere from other gaseous pollutants, particularly sulfur dioxide, nitrogen oxides, ammonia, and volatile organic compounds. Particulate matter is present in the atmosphere from a variety of sources (both natural and anthropogenic) that include emissions from motor vehicles, fragmentation of vehicles tyres and resuspension of road dust, power generation, metal processing, industrial combustion and smelting (Brook et al., 2004). Other sources are from agriculture, forest fires, residential wood burning, construction and demolition activities, pollens and moulds, windblown soil and burning of agricultural debris, sea spray and volcanic emissions. Particulate matter is made up of a lot of chemicals but the common constituents include nitrates, elemental and organic carbon, sulphates, organic compounds (e.g. polycyclic aromatic hydrocarbons), biological compounds (e.g. endotoxin, cell fragments), and a variety of metals (e.g. nickel, iron copper, vanadium and zinc.

Particulate matter is measured and regulated based primarily on mass within different size ranges because of its complex nature. In 1987, there was a shift in regulatory focus from total suspended particles to particles that could readily penetrate and deposit in the tracheobronchial tree, or PM_{10} (particulate matter with an aerodynamic diameter of $<10\mu$m). The USEPA regulated 24-hour and annual average standards for $PM_{2.5}$ (particulate matter with median aerodynamic diameter $<2.5\mu$m) in 1997, comprising the size fraction that can reach the small airways and alveoli. The function of the alveoli is to exchange oxygen and carbon dioxide molecules to and from the blood stream. $PM_{2.5}$ derives mostly from combustion sources and includes primary and secondary particles. On the contrary, PM_{10} (the coarse fraction) originates

mostly from natural sources such as crustal material (including windblown soil) and grinding processes (Brook et al., 2004). Other airborne particles (e.g. fungal spores, endotoxins and pollen grains) are found mostly in the coarse fraction (and larger particles), although both endotoxin (an essential component of the cell wall of gram-negative bacteria) and the antigenic protein content of pollen grains can also adsorb onto the surface of fine particulate matter. Larger particles (PM_{10}) exhibit a greater fractional deposition in the upper respiratory regions, whereas smaller particles (e.g. $PM_{2.5}$) show greater deposition in the lower respiratory tract and deep in the lungs. While $PM_{2.5}$ largely acts as a regional pollutant, there can be significant small-scale partial variability due to point source emissions (e.g. a smelter) or features such as street canyons in large cities as well as prevailing wind patterns that can affect human exposures (Brook et al., 2004).

Most recently, research on ultra-fine particles (UFPs) < 0.1μm in diameter, which results from combustion processes has been given considerable attention. UFPs cluster and combine and demonstrate a very high deposition in the alveoli. UFPs also make up a major portion of the actual particles within particulate matter. Because of its characteristic high surface area to mass ratio, UFPs potentially have enhanced biological toxicity. Ultra-fine particles may have the potential to be absorbed directly into the circulatory system, which would allow them to be disseminated systemically (EPA, n.d.).

2.6.2 Nitrogen Oxides

These are reactive substances known to include nitric oxide (NO), nitrogen dioxide (NO_2) nitrogen trioxide, and other oxides of nitrogen that are commonly referred to as "NO_x". A major source of particulate nitrate is gaseous nitric acid (HNO3), which is formed when NO_2 reacts with hydroxyl radicals during the day and when N_2O_5 reacts with water vapour at night (USEPA, 1993). Most toxicological and epidemiological research has focused on NO_2 because of the fact that it is one of the regulated air pollutants for which worldwide standards guidelines are available. Moreover, Nitrogen oxide from vehicular emissions and power plant is largely converted to NO_2, and plays a primary role in the formation of tropospheric ozone (O_3). NO_x is present in the atmosphere principally through the anthropogenic means of emissions from motor vehicles and industrial processes, especially in the generation of power. NO_2 is formed from the high temperature combustion and the oxidation of atmospheric N_2, and NO (Brook et al., 2004).

Local NO_x concentrations can be high near busy streets as a result of motor vehicle emissions. High volume of traffics contributes to the typical daily low background concentration of NO_x pattern with peaks usually in the morning and late afternoon. Under oxygen-rick combustion conditions, nitrogen in fossil fuels can be oxidized to nitrogen dioxide. NO_2 and NO are both formed naturally as a result of bacterial metabolism of nitrogenous compounds and, to a lesser extent, from fires, volcanoes and fixation by lightning. Tropospheric ozone and other photochemical oxidants are generated when NO_2 reacts with sunlight, whereas NO acts as an ozone scavenger (Lipsett, 2001). Humans can also be significantly exposed to Nitrogen oxides in non-occupational indoor settings (Spengler et al., 1994). Primary indoor sources of NO_x include unvented furnaces and stoves, kerosene space heaters and cigarette smoke (LEADERER, 1982). For urban settings, infiltration of ambient NO_2 from vehicular emissions may also influence indoor exposures (Spengler et al., 1994).

2.6.3 Carbon Monoxide

Carbon monoxide (CO) is almost an ever-present product of incomplete combustion of carbon-containing fuels. Common sources in the outdoors include emissions from motor vehicles, lawnmowers, engines on motorboats, chain saws and other devices that require fossil fuel combustion; residential wood-burning, improperly adjusted gas-burning and oil appliances; coal combustion and tobacco smoking (Hampson, 1992). In urban settings, the contributions of diesel and stationary source combustion are relatively small in comparison to gasoline powered engines (Harrison, 1999).

Carbon monoxide is an odourless, colourless and tasteless gas that binds to hemoglobin with an affinity 250 times that of oxygen, thereby interfering with the systemic delivery of oxygen to tissues. In addition, binding of carbon monoxide to hemoglobin causes an allosteric change in the conformation of the oxyhemoglobin complex that increases the oxygen affinity of the remaining binding sites and interferes with the release of O_2 at the tissue level (Brook et al., 2004). In the United States, the current ambient carbon monoxide concentrations suggest that in most circumstances, carbon monoxide serves more as an indicator of combustion-related activities than as a direct toxicant. Though, in some occasions (e.g. insufficiently ventilated parking structures), carbon monoxide could reach concentrations sufficient to lead to significant atherosclerotic disease or other cardiac conditions

2.6.4 Sulphur Dioxide

Sulphur dioxide (SO_2) is a colourless, highly irritating soluble gas with a pungent odour and taste. When it comes into contact with water, it forms sulphurous acid, which accounts for its strong irritant effect on eyes, skin and mucous membranes. Concentrations of ambient SO_2 are very low, in the range of 1 ppb without the addition of anthropogenic sources. The primary sources of SO_2 in the ambient air include combustion of sulphur-containing fuels, especially in power plants and diesel engines and roasting of metal sulphide ores. Sulphur dioxide is oxidized to sulphur trioxide, which, because of its strong affinity for water, can be rapidly hydrated to form sulphuric acid (WHO, 1987). Increased levels of sulphur dioxide have been associated with widespread illnesses in several 20^{th} century air pollution catastrophes; however, much of the illnesses and deaths in these episodes may have been due to its role in the formation of particulate sulphates. Even though SO_2 concentrations is generally lower indoors than outside, the use of kerosene space heaters can give rise to substantial indoor SO_2 concentrations (LEADERER, 1982).

2.6.5 Ozone

Ozone (O_3) is not emitted directly into the atmosphere but is created through natural processes and by anthropogenic actions.O_3 is composed of three oxygen molecules joined together with the two basic oxygen molecule (O_2) and an additional third atom which makers ozone an unstable highly reactive gas. It is created when emissions from vehicles and industries containing oxides of nitrogen (NOx) and volatile organic compounds chemically react in the presence of sunlight ("Ground-level Ozone Basics | US EPA", n.d.). It is a colourless-to-bluish gas with a characteristic odour similar to that of electrical discharges. Low level exposure is ubiquitous, because it is more readily formed during the summer months and is usually at the highest concentrations in the afternoon or early evening. In the stratosphere ozone is formed by the action of solar radiation on molecular oxygen (O_2). Stratospheric O_3 prevents high-energy ultraviolet radiation from entering the atmosphere and many terrestrial life-forms would be unable to survive without the stratospheric O_3 layer.

Since the 1950s, ozone has been recognized as the major element in the formation of photochemical smog. In the troposphere, it is also known as ground-level ozone and is formed by the action of solar UV radiation on nitrogen oxides and reactive hydrocarbons. Nitrogen oxides and reactive hydrocarbons are from emissions from motor vehicles, power plants, industrial boilers, refineries, chemical plants and other industrial sources. The reaction sequence for the formation of O_3 involves photolysis of nitrogen dioxide (NO_2) to NO and oxygen atoms. Under desirable circumstances, there is minimal buildup of O_3. However, volatile organic compounds (VOCs) can facilitate the oxidation of NO to NO_2 by alternative mechanisms. These reactions reduce NO scavenging, which allows O_3 concentrations to increase (Brook et al., 2004).

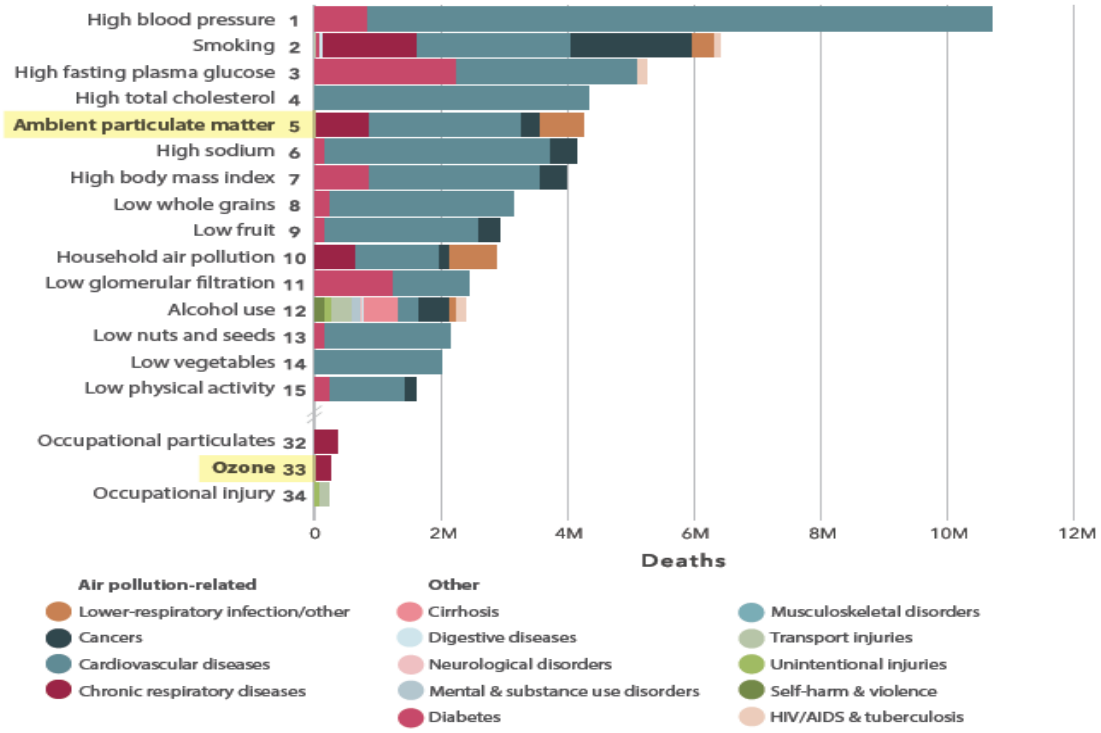
On hot sunny days in urban environments, ozone is most likely to reach unhealthy levels but can also reach high levels during colder months. Ozone can also be transported long distances by wind, so even rural areas can experience high ozone levels. The typical formation of ground-level ozone in populated areas is characterized by a broad peak that lasts from the late in the morning until late in the afternoon or early evening (USEPA, 2017). In addition, the increase in ozone concentrations in remote areas that are far away from primary sources can be by means of large-scale transport moving into these areas. Factors affecting the temporal O_3 patterns include wind speed and direction, temperature inversion, addition of other O_3 precursors so that peak concentrations may occur anytime from noon until late in the evening (Lioy & Raymond V., 1989). Human activities are major sources of O_3 precursors, although there are also natural sources of ozone precursors which includes the intrusions on stratospheric O_3 (USEPA, 1996).

2.7 Global Burden of Ambient air pollution

The Global Burden of Diseases, Injuries and Risk Factors study in 2015 (GBD 2015) estimated the burden of disease attributable to seventy-nine risk factors in 195 countries from 1990 to 2015. The study identified air pollution as a leading cause of global disease burden, especially in low-income and middle-income countries. Ambient air pollution in particular particulate matter with an aerodynamic diameter of $<2.5\mu$m was identified as a leading risk factor for global disease burden with an estimated 2.9 million attributable deaths in 2013. An additional 217,000 deaths were attributable to long-term ozone exposure (Collaborators, 2017).

According to (Cohen et al., 2017), ambient air pollution contributes substantially to the global burden of disease in 2015. This burden of disease has increased for the past 25 years (1990-2015) due to population ageing, increasing non-communicable disease rates and the increasing air pollution in low-income and middle-income countries. In addition, the global exposure to air pollution and its disease burden as shown in Fig 6 illustrates that the air pollutant $PM_{2.5}$ was the fifth highest ranking risk factor for death in 2015. Particulate matter with an aerodynamic diameter of $<2.5\mu$m was responsible for 4.2 million deaths from heart disease and stroke, lung cancers and respiratory illnesses. Ground level ozone was ranked as the 33rd risk factor causing deaths and was responsible for an additional 254,000 deaths.

Table 3: Global ranking of risk factors for total deaths from all causes for all ages and sexes in 2015
Source: State of Global Air-2017



Moreover, recent reviews by the USEPA and WHO have shown that the long-term exposure to ambient air pollution is responsible for increased mortality and morbidity from respiratory and cardiovascular diseases, lung cancer and shortens life expectancy.

2.8 Health Impacts of Ambient Air Pollution

Ambient air pollution is a major cause of death and disease globally. The health effects range from increased hospital admissions and emergency room visits, increased mortality, to increased risk of premature death (Brunekreef & Holgate, 2002). Premature deaths linked to ambient air pollution are mainly from heart disease, stroke, chronic pulmonary disease, lung cancer and acute respiratory illnesses among children. The World Health Organization estimates that worldwide ambient air pollution accounts for

- 29% of all deaths and diseases from lung cancer
- 17% of all deaths and disease from acute lower respiratory infection
- 24% of all deaths from stroke
- 25% of all deaths and disease from ischaemic heart disease
- 43% of all deaths and disease from chronic obstructive pulmonary disease.

Pollutants of interest and with the strongest evidence for public health concern include particulates with an aerodynamic diameter $<10\mu$m (PM_{10}) and particulates with an aerodynamic diameter $<2.5\mu$m ($PM_{2.5}$), ozone (O_3) sulphur dioxide (SO_2) and nitrogen dioxide (NO_2).
The health risks associated with particulate matter is well documented in terms of mortality and cardiovascular and respiratory effects. It is also used widely as an indicator to assess the health effects of exposure to ambient air pollution. Because of its small size PM_{10} can penetrate and be deposited in the upper respiratory tract. On the other hand, $PM_{2.5}$ can penetrate further deep into the gas exchange region of the lungs (alveoli) and cause respiratory and cardiovascular impacts (Kampa & Castanas, 2008). Because of its toxicity and ability to penetrate deep into the respiratory system, particulate matter has been classified as a cause of lung cancer by the WHO's International Research Agency for Research on Cancer (IARC).

The relationship between particulate matter and lung cancer was also observed in the study by (C.A.P, 2002) fine particulate and sulphur dioxide related pollution were associated with all-cause, lung cancer and cardiopulmonary mortality. They found that each 10-μg/m^3 elevation in fine air particulate pollution was associated with about a 4%, 6%, 8% increased risk of all-cause, cardiopulmonary and lung cancer mortality respectively. Moreover, they concluded that long term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. Similar findings was made in the cohort of never-smokers by (Turner et al., 2011) where the researchers observed that each 10μg/m^3 increase in $PM_{2.5}$ concentrations was associated with a 15-27% increase in lung cancer mortality. Turner and other concluded that their findings has contributed and strengthened the evidence that recent ambient concentrations of $PM_{2.5}$ are associated with small but measurable increases in lung cancer mortality.

Both short-term and long-term exposure to ambient air pollution can lead to reduced lung function, respiratory infections and aggravated asthma in both children and adults. This was observed in a cohort study by (JAMESGAUDERMAN et al., 2000) where after appropriate adjustments found that ambient air pollution was correlated with statistically significant decreases in lung function. James Gauderman and others came to the conclusion that these finding suggests that exposure to air pollution may lead to a reduction in maximal lung function which occurs early in adult life, and ultimately to increased risk of chronic respiratory illnesses in adulthood. These findings were similar to the European birth cohort study by (Gehring et

al., 2013) where a 20 $\mu g/m^3$ increase in levels of NO_2, Nitrogen oxides, and a $5\mu g/m^3$ of $PM_{2.5}$ was associated with small decreases in lung function. The authors of the European birth cohort study deduced that exposure to air pollution may result in reduced lung function in schoolchildren.
On the effects of air pollution on asthma, a prospective cohort study by (Jerrett et al., 2008) found significant associations between incident asthma and exposure to ambient NO_2. In addition, the risks observed in this study suggest that air pollution contributes to the new onset of asthma. In addition, in the time series study by (Schwartz, Slater, Larson, Pierson, & Koenig, 1993), they observed a significant association on the daily counts of asthma emergency room visits for persons under 65 with PM_{10} exposure on the previous day. But, Schwartz and others deduced from their study that the mechanisms triggering or exacerbating asthma remains unclear. However, a study by (Halonen et al., 2008), observed after accurate particle size segregation was used, nucleation mode was associated with asthma visits of children. Moreover, Halonen and others also observed a positive association for the pooled asthma-COPD visits with $PM_{2.5}$. This study by Halonen et al., also found that coarse and accumulation mode particle levels was statistically significantly associated with the pooled asthma-COPD visits. Halonen and others concluded that smaller particle size (<250nm), gaseous air pollutants and traffic related $PM_{2.5}$ (at longer lags) were significantly associated with hospital emergency room visits for asthma among children. In contrast, $PM_{2.5}$ coarse particles and gaseous pollutants had a more immediate effect on the pooled asthma-COPD visits of the elderly.

Furthermore, the World Health Organization suggests that maternal exposure to ambient air pollution is associated with adverse birth outcomes such as low birth weight (LBW), pre-term birth and small gestational age (SGA) births. This was observed in a meta-analysis study by (Dadvand et al., 2012) where the authors found that term low birth weight was positively associated with a $10\mu g/m^3$ increase in PM_{10}, OR = 1.03 (95% CI:1.01, 1.05) and $PM_{2.5}$, OR = 1.10(95% CI:1.03, 1.18). Dadvand and others came to the conclusion that their combined effect estimates study supports an adverse impact of maternal exposure to particulate pollution on fetal growth. In addition, a population based study by (Maroziene & Grazuleviciene, 2002) found that the risk of preterm birth increased by 25%, adjusted OR = 1.25 (95% CI: 1.07-1.46). Maroziena and Grazuleviciena concluded that a relationship exist between NO_2 exposure and the risk of preterm birth. Another study by (Malmqvist et al., 2011), they observed a statistically significant association between small for gestational age (SGA) births and both nitrogen oxides (NO_x) and traffic density. In their subgroup analysis, Malmqvist and other observed an increased risk of SGA for girls, OR = 1.12(95% CI: 1.01-1.24); they also observed increased risk among mothers who had not changed residency during pregnancy, OR = 1.09 (95% CI: 1.01-1.18). In this study the authors concluded that controlling for confounders is crucial, especially those that are linked to socioeconomic and spatial gradients.

According to the World Health Organization (WHO, 2019), there is emerging evidence that suggests ambient air pollution may affect diabetes and neurological development in children. This relationship between air pollution and diabetes was observed in a cohort study by (Raaschou-Nielsen et al., 2012)whereby the authors found a statistical significant association between long-term exposure (exposure to above $19.4\mu g/m^3$ - upper quartile) to traffic-related air pollution at the residence and diabetes mortality, mortality rate ratio, MRR of 2.15 (95% CI 1.21, 3.83). These findings were consistent with the meta-analysis study by (Janghorbani, Momeni, & Mansourian, 2014) whereby they found that exposure to air pollution was associated with slight increase risk of diabetes and susceptibility of people with diabetes to air pollution. Moreover, the birth cohort study by (Guxens et al., 2012) observed the effects of exposure to air pollutants on infant mental development. Guxens and others concluded that their findings suggests that prenatal exposure to residential air pollutants may

adversely affect infant mental development, but potential effects may be limited to infants whose mothers report low antioxidant intakes.

Even though all populations are affected by air pollution, the burden of disease is inequitable within and across countries. Most of the disease burden is evident in low-and middle-income countries including the poor and marginalized population. To some extent, this is due to the intense process of urbanization and industrial development in a very short period of time and this phenomenon has had deleterious effects on the health of the people living in these countries (Mannucci & Franchini, 2017). These populations tend to inhabit locations near busy roads and industrial sites characterized by high levels of outdoor air pollution.

2.8.1 Ambient air quality and cardiovascular disease

The cardiovascular system is also known as the circulatory system and includes the heart, arteries, veins capillaries and blood. These vital structures are critical in the process of pumping deoxygenated blood to the lungs for gas exchange as well as pumping oxygenated blood to the body's tissues to support their metabolic functions. The cardiovascular system has three major functions and includes:

1. Transportation of materials - the cardiovascular system transports blood to almost all of the body's tissues. The blood delivers nutrients and oxygen and removes wastes and carbon dioxide to be processed or removed from the body.

2. Protection from pathogens - this is another of its function and it protects the body through its white blood cells. White blood cells clean up cellular debris and fight pathogens that have entered the body. Platelets and red blood cells form scars to seal wounds and prevent pathogens from entering the body and liquids from leaking out. Blood also carries antibodies that provide specific immunity to pathogens that the body has previously been exposed to or has been vaccinated against (Innerbody, 2016).

3. Regulation of the body's homeostasis - the cardiovascular system is instrumental in the body's ability to maintain homeostatic *(maintaining internal stability owing to the coordinated response of its parts to any situation or stimulus that would tend to disturb its normal condition or function)* control of several internal conditions. Blood vessels help maintain a stable body temperature by controlling the blood flow to the surface of the skin. Blood vessels near the skin's surface open during times of overheating to allow hot blood to dump its heat into the body's surroundings. In the case of hypothermia, these blood vessels constrict to keep flood flowing only to vital organs in the body's core. Blood also helps balance the body's pH due to the presence of bicarbonate ions, which act as a buffer solution (Innerbody, 2016).

Many serious conditions and diseases can cause our cardiovascular system to stop working properly. It is now well reasonably well established that both short-term and chronic air pollution exposures are related to cardiovascular diseases (Brook et al., 2004). Ever since the late 20th Century, epidemiological and clinical evidence has led to an increased concern about the potential harmful effects of ambient air pollution and its association with heart disease and stroke. Of special interest are common air pollutants that include particulate matter with aerodynamic diameter $<10\mu$m (PM_{10}) and aerodynamic diameter <2.5 ($PM_{2.5}$), carbon monoxide, oxides of nitrogen, sulphur dioxide, ozone and lead (Brook et al., 2004).

The most common route for these pollutants to enter the cardiovascular system is via inhalation. When inhaling air travels from the upper respiratory tract which includes the nasal cavity,

pharynx, epiglottis and larynx to the lower respiratory tract consisting of the trachea, bronchi, bronchioles and lungs. Of all the pollutants that pass through the respiratory system (refer to Fig. 7), particulate matter with an aerodynamic diameter of <2μm and <0.1μm have the ability to penetrate the lung alveoli (where gas exchange of oxygen and carbon dioxide occur) and enter the blood stream whereby it exerts the adverse health effects (Franck et. al., 2011). Because of its very small size, most particulates with an aerodynamic diameter of 2μm to 10μm are deposited in the nasal cavities and upper airways. Particulat matter has been recognized as being associated with inducing adverse health effects because it may contain substances that can be transported to the respiratory tract (Lee, Kim, & Lee, 2014).

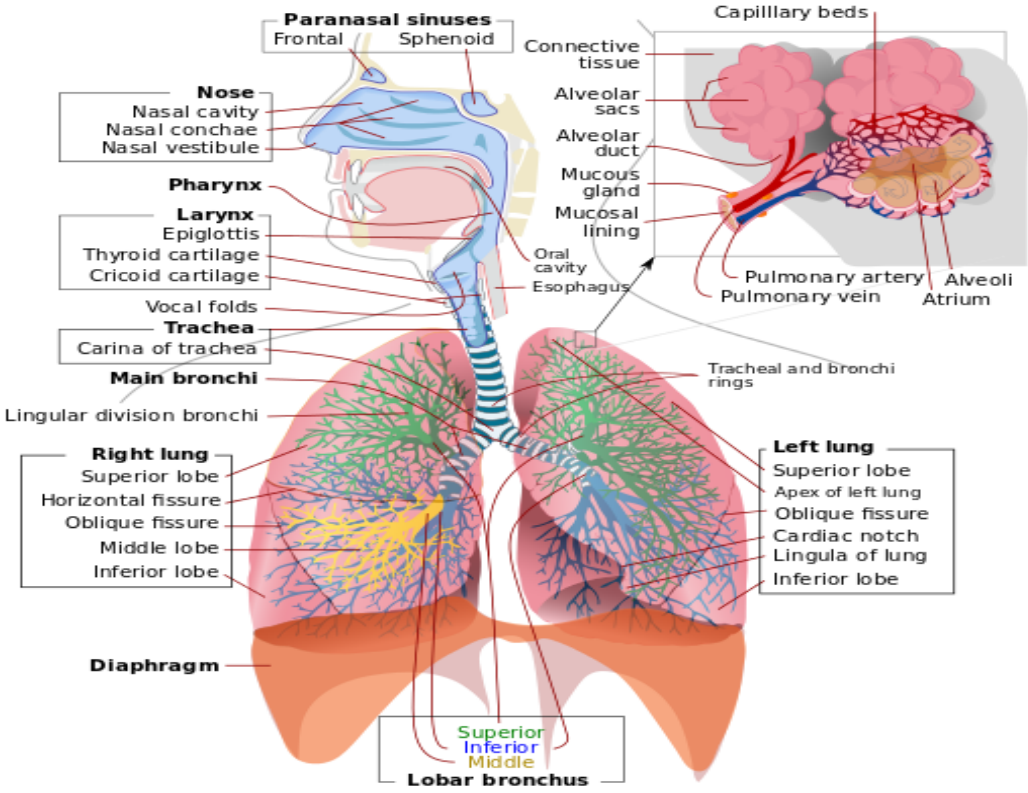


Figure 5: The respiratory system showing the upper and lower respiratory tract as well as the alveoli. Source: US National Library of Medicine

There is a growing body of epidemiological knowledge and studies that the greatest health threat due to air pollution is cardiovascular disease (Lee, Kim, & Lee, 2014). Because of its minute size, particulate matter can be inhaled deep into the lungs, with a portion depositing in the alveoli and entering the pulmonary circulation and apparently the systemic circulation (Sun, Hong, & Wold, 2010). Inhalation of particulate matter or ultrafine particles (UFPs) triggers inflammatory responses in the lung and increases the release of inflammatory mediators in the blood. This in turn can lead to various changes in the cardiovascular system, such as an increase in blood coagulability and the progression of atherosclerotic lesions (Nakane, 2011).

Studies from around the world have consistently shown that both short- and long-term exposures to pollutants, in particular Particulate Matter are associated with a host of cardiovascular illnesses including, myocardial ischaemia and infarctions, heart failure, arrythmias, strokes and increased cardiovascular mortality. Moreover, evidence from cellular/toxicological experiments, controlled animal and human exposures have demonstrated

several mechanisms by which pollutant exposure may both trigger acute events as well as prompt the chronic development of cardiovascular diseases (Brook, 2008). In a series of epidemiological studies,(Dockery et al., 1993) and (Samet, Dominici, Curriero, Coursac, & Zeger, 2000) established that air pollution have adverse effects on the cardiovascular system and that brief exposures to air pollution have been associated with increased cardiovascular related morbidity and mortality. This pathology link have particular implications in low- and middle-income countries. These low- and middle-income countries are rapidly developing, hence, air pollution concentrations are continuing to rise. Moreover, the study by (Barnett et al., 2006) on the effect of air pollution on hospitalizations for cardiovascular disease in elderly people in Australia and New Zealand cities found that particulate matter (PM_{10} & $PM_{2.5}$), NO_2, SO_2 and CO were significantly associated with higher admissions amongst the elderly (≥65yrs) than the younger age group (15-64 yrs). The findings from this study showed that the elderly admissions increased for total cardiovascular disease (2.2%), all cardiac disease (2.8%), cardiac failure (6%), ischaemic disease (2.3%) and myocardial infarction (2.9%). The authors of this study in New Zealand and Australia concluded that their advanced age, frailty and with probably pre-existing heart conditions made the elderly population vulnerable. Interestingly, these associations were found at concentrations that were below normal air quality health guidelines and the authors suggest that these guidelines have to be revised and lowered if possible to improve cardiovascular health amongst the vulnerable population. Similar findings on the effects of pollutants on cardiovascular health of the elderly population were also found in the study by (Liu et al., 2015)

2.8.2 Ambient air quality and Acute Myocardial Infarction

Acute myocardial infarction is also referred to as heart attack, acute coronary syndrome or heart infarction and is a life-threatening condition. It occurs when blood flow to the heart muscle is abruptly cut-off, causing tissue damage. This is usually the result of a blockage in one or more of the coronary arteries. A blockage can develop due to build-up of plaque, a substance mostly made of fat, cholesterol and cellular waste products. Epidemiological studies for the past two decades have consistently shown that the level of air pollutants in the ambient air have been associated with hospitalizations due to acute myocardial infarction. For instance, the epidemiological study by (Peters et al., 2001) examined the effects of short-term exposure to fine particulate matter and risk of acute myocardial infarction in the greater Boston area, Massachusetts, USA. In their case-crossover study, Peters and colleagues observed that an increase in the levels of fine particulate matter ($PM_{2.5}$) in the previous 2 hours was associated with an increased risk of myocardial infarction (MI) onset. They also observed a significant association between high 24-hour average concentrations of fine particulate matter and an increased risk of MI with a 24-hour delay. From these findings Peters and other suggested that increased concentrations of fine particulates in the air may elevate the risk of myocardial infarction after a few hours to a day of exposure.

In another case-crossover study by (Rasche et al., 2018), the authors examined the effect of the rapid changes in the levels of NO_2, O_3, PM_{10}, oxides of nitrogen (NO_x) and risk of myocardial infarction for the populace in the city of Jena, Germany. Rashe and colleagues observed that a 20 $\mu g/m^3$ and 8-20$\mu g/m^3$ increase in NO_x was associated with up to 121% increased risk of MI with a lag time (Lag-2 days and Lag-3days). In addition, rapid changes in NO_2 (lag-1day) was associated in a close-to-linear risk of myocardial infarction. Moreover, increases in NO_2 by more than 8$\mu g/m^3$ increased the risk of MI by 73%. In this study, PM_{10} concentrations was not associated with any increased risk in MI. From these findings, Rasche and others suggested that the risk of Myocardial Infarction was not only dependent on short-term or long-term exposure to gaseous pollutants but also in the dynamic and magnitude of their increases.

In a similar study design to those discussed above, (Collart et al., 2015) investigated the association between short-term exposure to air pollutants and the triggering of acute myocardial infarction (AMI) in Charleroi, a heavily polluted region in Wallonia, Belgium. Collart and colleagues observed that the risk of AMI increased when, (1) PM_{10} and NO_2 concentrations increased by $10\mu g/m^3$ and, (2) when there was a 1^oC increase in temperature. From these findings Collart and others concluded that the effects of the short-term effects of air pollutants and the risk of acute myocardial infarction was stronger in the warm period.

All the three studies discussed here employed the case-crossover (Maclure, 1991) study design and all showed that the risk of triggering acute myocardial infarction can occur in both short-term and long-term exposure to air pollutants.

2.8.3 Potential Biological Mechanisms

The general pathway that is associated with air pollution causing cardiovascular events includes direct and indirect effects of pollutants on the cardiovascular system, respiratory system and blood. The biological mechanism by which these effects are mediated is known as pulmonary oxidative stress or inflammatory responses. Pulmonary oxidative stress is an imbalance between the production of free radicals and the ability of the body to counteract or detoxify their harmful effects through neutralization by antioxidants (Brook et al., 2004). Direct effects may occur by way of agents that readily cross the pulmonary epithelium in to the circulation, such as gases and perhaps ultra-fine particles (NEMMAR et al., 2001) along with soluble constituents of particulate matter with an aerodynamic diameter < 2.5 microns ($PM_{2.5}$), e.g. transition metals. Pulmonary neural reflexes are activated after particulate matter interactions with lung receptors and this may play a role. Subsequent changes in autonomic tone, under favourable circumstances might contribute to the instability of a vascular plaque or initiate cardiac arrhythmias. These direct effects of air pollution indicate a probable explanation for the occurrence of rapid (within a few hours) cardiovascular responses, such as increased myocardial infarctions (Peters, Dockery, Muller, & Mittleman, 2001). Less acute (several hours to days) and chronic indirect effects may occur via pulmonary oxidative stress/ inflammation induced by inhaled pollutants. Later on, this may contribute to a systemic inflammatory state, which may in turn be capable of activating haemostatic pathways, impairing vascular function, and accelerating atherosclerosis (a disease in which the inside of an artery narrows due to buildup of plaque)(Brook et al., 2004). A general scheme showing potential mechanisms of the effects of ambient air pollutants (in particular, particulate matter) on the cardiovascular system is shown in Figure.

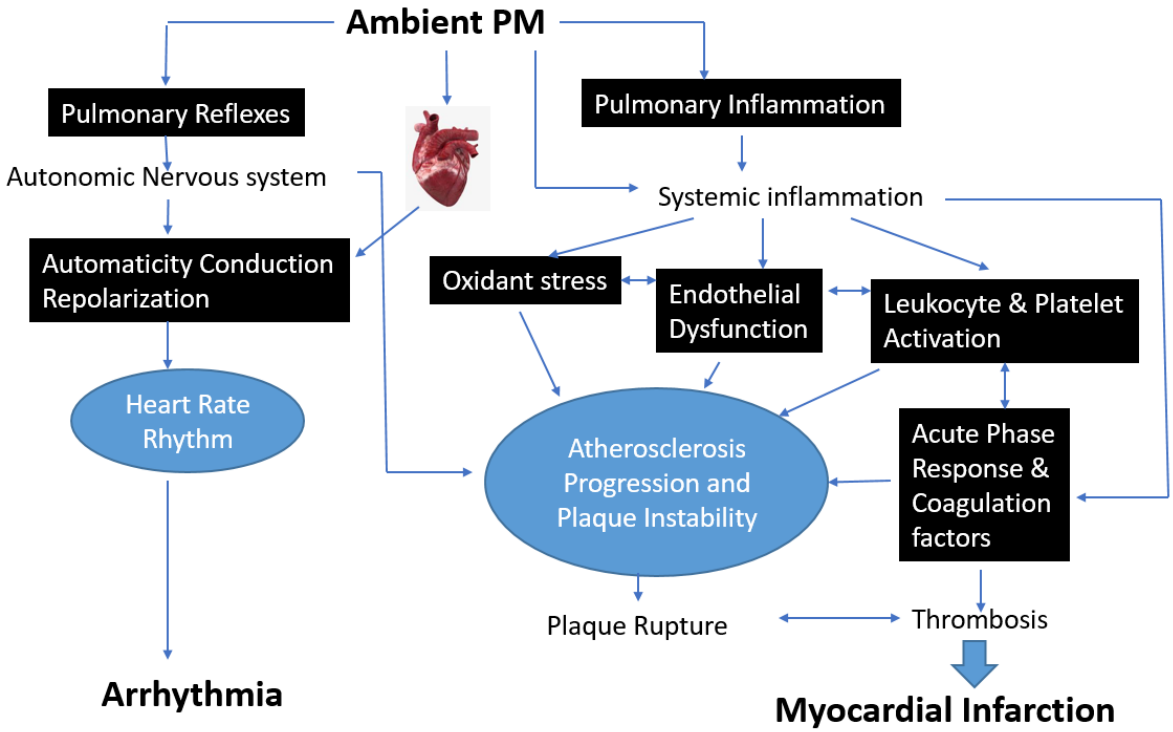


Figure 6: Illustrates the possible biological mechanisms linking particulate matter with acute myocardial infarction. Adapted from Brook et al., 2004

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CHAPTER 3

METHODOLOGY

3.1 Summary

This chapter has thoroughly deliberated on the methodologies that was used during the course of this study. Initially, this chapter has addressed the first research aim of this study. The first aim was collecting data on the ambient air quality in the Lautoka Central Business District for a period of three months. The steps and procedures taken to collect air quality data has been thoroughly explained herein. The study area and its description is highlighted with pictures to view the significance and characteristics of the chosen sites. Air quality instruments used and its setup also explained with pictures to view how the air quality monitoring sensors were mounted on to the camera poles. Data collection, processing and analysis methods has been thoroughly described. Secondly, a systematic review and meta-analysis was conducted to assess the association between air quality and heart disease. Explanation on the systematic review and steps for the meta-analysis and how the outcome was pursued is also discussed.

3.2 Introduction

This chapter describes the methods used for this study. A brief of the procedures involved in the collection of the ambient air quality and weather data will be presented. The processes involved in manipulating (cleaning and processing) the raw dataset before it is being described and analyzed will be discussed. The systematic review and meta-analysis procedures will also be presented. In essence, it will prudent to note that as a recipient of the New Zealand Scholarship, 3 months was the maximum allowable time allocated for master's scholars to conduct home-based research.

3.3 Study design

This study employed a combination of collection of field data on air quality by measuring criteria pollutants (excluding lead) for a period of 3 months, meta-analysis and developing a dose-response model of the association between criteria pollutants and risk of acute myocardial infarction hospitalizations. Pooled estimates from the meta-analysis with the air quality data was sued to determine the risk of AMI hospitalization in Lautoka. Lead was not included in this study simply because of the parameter. Essentially, collection air quality data was for a maximum period of 3 months in order to conform to the NZ scholarship allocated time for master's scholars to conduct home-based research.

Hence, the case-crossover study design proposed by was the choice to examine the transient effects on the risk of acute events. This study design is being widely used in recent epidemiological studies to evaluate the short term effects of exposure to air pollutants on respiratory disease and cardiovascular events. Only cases are used and comparisons of an individual's exposure experience are made just before an event with exposure at other time periods of interest. An advantage of case-crossover design is that each case serves as its own control and confounding risk factors that are invariant and slowly changing such as gender, age, race, socio-economic status and smoking is controlled for by the design itself.

3.4 Study site identification and description

Air quality data was collected within the Central Business District (CBD) of Lautoka City. The Chief Executive Officer of the Lautoka City Council (LCC) granted permission to conduct the study within the boundaries of Lautoka CBD as well as the utilization of the council's resources. These resources includes a 4 ancillary staff, a cherry picker vehicle, custom made steel brackets to affix the air quality monitoring sensors to designated steel poles and other required tools. A senior Environmental Health Officer was designated to guide the student because of his vast knowledge of the city and to locate the best possible locations to mount the monitoring sensors. The sites were chosen with guidance from the LCC staff who have firsthand experience of the vehicle movement in the city. In essence, the study had to gather relevant data with respect to sections of the city where, (1) always an influx of people on a daily basis as well as (2) high rate of vehicle movement. Three sites were initially selected within the CBD area to allow collection of ambient air from three separate but distinct locations. However, only two AQMS was used as the third one developed problems prior to being used.

Figure 7: Shows the boundaries of the Lautoka CBD. Source: Google Maps

The two sites according to the senior Environmental Health Officer and LCC city workers are generally high volume used roads by all vehicles, workers and pedestrians on a daily basis. Site 1 as shown in Figures 9 & 10 was chosen because of the volume of traffic and usage of this road by daily commuters into the city. This air quality monitoring site is at the juncture of Vitogo Parade (highlighted in yellow in Fig 8) and Narara parade. The traffic management plans for this city does not allow buses to use this section of the city in order to streamline the traffic flow within the city. Adjacent to this site are major clothing retail stores and on the other side is a public park for community recreational activities. A railway runs parallel to the Vitogo parade and is used to cart sugar cane from farms located in the outskirts of Lautoka City and from Nadi town. Site 2 is located at the junction of Tukani Street and Vakabale Street as shown in Figures 11, 12 & 13. Traffic volume along this site is always high especially for buses and heavy trucks. Adjacent to Site 2 are restaurants and retail stores as well as the main bus station for the city. The public market is about 30m from this monitoring site and the density of people around this area on a daily basis is always high. This site is at the intersection of a T-junction with vehicles including small vehicles, trucks and buses turning in and out.

Figure 8: Mounting of the Air quality transmitter on site 1 along Narara Parade (highlighted in yellow in Fig 7) with a view of its immediate surrounding

The Air Quality Monitoring Sensors (AQMS) are being prepared to be placed in Site 1. The Lautoka City Council electricians and ancillary staff can be seen here with their resources to assist in the mounting of the study equipment onto the camera steel pole.

Figure 9: Mounting of the air quality transmitter completed for site 1. On the background is a view of the immediate surroundings for the site

The immediate surroundings of Site 1 can be seen in Figures 8 and 9. No buses are allowed to transit along these road (Narara Parade). The picture on the left shows the final setup of the AQMS and other relevant components.

Figure 10: A view of site 2 and its immediate surroundings. This site is at the center of an intersection

Figure 10 shows the immediate surrounding of Site 2 and the locale for the AQMS. Vehicles allowed to transit along these roads includes all light and heavy vehicles including buses.

Figure 11: Another view of site 2 with the LCC staff assisiting in assembling the AQMS

This figure shows immediate vicinity of site 2 as well as the Lautoka City Council electricians and ancillary staff assisting in the setting up of the study equipment before it is being placed on the camera pole.

Figure 12: Lautoka city council electricians and ancillary staff assisting with the mounting of the air quality transmitters

The final touches in mounting the air quality monitoring sensors to the camera pole on Site 2 with the able assistance of the Lautoka City Council workers.

3.5 Data collection

Ambient air quality was measured for a period of three months from 15th August 2018 to 15th November 2018. The VAISALA Air quality transmitter (AQT) 400 series which consists of two products (AQT 420 & AQT 410)the AQT 420 was the equipment of choice to collect air quality and weather data as it measured most of the common air pollutants (PM10, PM2.5, SO2, NO2, O3, CO) excluding lead and weather variables (Temperature, relative humidity, atmospheric pressure). This is one of the latest air quality monitoring equipment that is accurate and uses high-precision instrumental methods in order to understand how contaminant levels fluctuate over short time periods (hours or days). One of the distinct features of this instrument is that it compensates for the impact of ambient air conditions and is specifically designed for urban areas, road networks or industrial sites and around transportation hubs (Vaisala 2018). Two air quality monitoring sensors were mounted on two existing traffic camera poles (refer to Figs 9 & 12). The instruments mounted on each poles includes a flexible solar panel, one VAISALA Air Quality Transmitter AQT 420 series, a box containing a 12v rechargeable alkali battery, a solar panel regulator and a serial data logger. The AQT 420 series was located at a height of 3m from the ground as a precautionary measure from vandalism and theft. Ambient air quality data on levels of particulate matter with an aerodynamic $<10\mu m$(PM_{10}) and aerodynamic $<2.5\mu m$ ($PM_{2.5}$), sulphur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO) and ozone (O_3) were collected at the two air quality monitoring sites. The pollutants PM_{10} and $PM_{2.5}$ was measured as microgram per cubic meter ($\mu g/m^3$) and the pollutants CO, NO_2, SO_3, O_3 were measured as parts per billion (ppb). The VAISALA Air Quality Transmitter 420 series also measures meteorology data like temperature in degrees Celcius (^0C), relative humidity in percentage (%) and pressure in hectopascal (hPa). Ambient air quality and meteorology data from the VAISALA Air Quality Transmitter 400 series was transferred into the serial data logger and then stored in a universal serial bus (USB) flash drive in text document (txt).

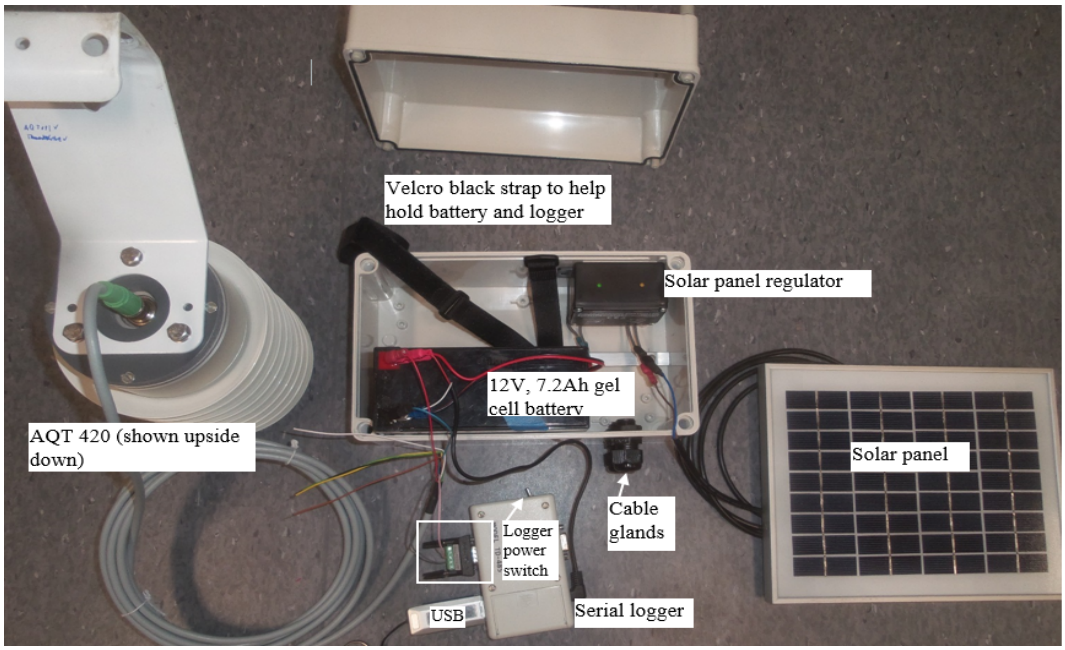

Figure 13: A close-up view of the components for the air quality transmitter 420

Figure 14: Approximate layout of the AQMS components prior to being closed and mounted to the pole

Once all connected up as shown in Fig.14, the logger is switched on and the power LED (light-emitting diode) will show up. The Data light (bright green) will flash once per minute as data is sent through.

3.6 Data Preprocessing

Air quality data was then exported into a Microsoft excel spreadsheet and rows of readings when the equipment was being tested at the University of Canterbury Geography lab were removed. Columns that had no relevance to the desired data set were removed as well. A new row was then inserted at the top of the excel sheet and used as the headings for each pollutant and meteorology variable. Columns A-K was then renamed to reflect corresponding parameters. After all these steps were taken to clean the raw data, a new excel spreadsheet was created and the cleaned data pasted onto it and given the name Site 1_data. These steps were replicated for the data obtained from Site 2 except that the first 6 rows was not removed. The cleaned data was then pasted onto a new excel spreadsheet and given the name Site2_data. On the completion of cleaning the raw data from both sites, a third spreadsheet was also created and named 'Master_Sheet'. This Microsoft excel sheet contains cleaned data from both Site 1 and Site 2 merged into one data set for the Lautoka city.

3.7 Data Processing

Now that the data is clean, it was imported into R Studio for further processing before beginning with the analysis. R Studio is a free and open-source integrated development environment for R, a programming language for statistical computing and graphics. R-Studio uses the graphical environment of the computer to facilitate interactions with R for example, it has a Console window for typing code, a data window for reviewing data frame and other data structures, a work space for viewing all of the data loaded into R, Plot area for showing visualizations and a tabbed window control to show other displays (RStudio, 2018). Processing data meant that I had to manipulate the clean data in order to impute the mean or average hourly and daily values of the environmental exposure variables which includes temperature, pressure, humidity, nitrogen dioxide, sulphur dioxide, carbon monoxide, ozone, particulate matter with an aerodynamic diameter of <2.5 microns and particulate matter with an aerodynamic diameter of <10 microns.

In R Studio, a new project was created and the dataset 'Master_Sheet.xls' with a total of 6,822 observations of 11 variables was imported into R and renamed '*aqdataset'*. The R packages (library tidyverse, library readxl and library lubridate) was loaded onto the R console as these packages will be used to process and later on analyze the dataset. These R packages makes it easier to work with dates and times and to read, write and format excel files. In addition, it is a coherent system of packages for data manipulation, exploration and visualization that share a common design philosophy. The '*aqdatset'* was corrected so that the year was separated from the months, months were separated from the days, days were separated from the hours, hours were separated from minutes and minutes separated from seconds. After the analysis of the above, 7 new variables were added to the existing dataset and the new dataset with a total of 6822 observation of 18 variables was renamed as. The new variables were year, month, day, hour, minute, second and date.
The final step in data processing was to group the data based on hour and then based on day. To do this the *group by* and pipe function was used to impute and summarize the mean values of all the observations. The outcome of this final data processing step yielded a total of 121 observations of 13 variables and the final dataset was named, *'aqdata_revised'*. Now that the final dataset has been imputed, the next step was out carry out the analysis. Please refer to Appendix 3 on the particulars of the R script used for data processing.

3.8 Data Analysis

3.8.1 Descriptive Statistics

The daily (24-hour) average or mean concentrations for the pollutants, particulate matter with an aerodynamic diameter <2.5 and aerodynamic diameter <10 microns, sulphur dioxide, nitrogen dioxide, zone and carbon monoxide was imputed using the formula and the values tabulated. Mean values for the weather variables, temperature, relative humidity and atmospheric pressure was also obtained. The range which included the minimum and maximum values for all these environmental variables was also obtained when imputing their mean values. Values for the 25^{th}, 50^{th} & 75^{th} percentiles were also acquired by arithmetic means and the value tabulated.

To measure the statistical relationship between the environmental variables (PM_{10}, $PM_{2.5}$, SO_2, NO_2, O_3, CO, temperature, relative humidity and atmospheric pressure) Pearson's correlation coefficient, *r*, was employed. Pearson's correlation coefficient is the best method of measuring the association between two variables of interest because it is based on the method of covariance. It provides information about the magnitude of the association, or correlation as well as the direction of the relationship.

3.8.2 Time Series

The time series approach was used to determine and construct the concentrations of the pollutants and the weather variables. Mean concentrations for days of the week (Monday to Saturday) were imputed and their values plotted. Note there are no mean concentrations for Sunday as this day was dedicated for data extraction from the data logger as well as checking for the power in the batteries. Similarly, concentrations for each hour was obtained and the mean imputed (i.e. concentrations for every 1 am of every day were obtained and the average imputed. This procedure was repeated for 2am, 3am, and 4am and so on). This was done for the whole 24 hours (0100hrs to 2400hrs) period in order to be able to visualize the observations of the variables against time. Visualizing the air quality and meteorology data enables the identification of the nature and distinct features of the observations. Time series analysis was done in the Microsoft Excel 2016 software program.

3.9 Meta-Analysis

This study used the meta-analysis method to combine and analyze quantitative evidence from primary studies examining the short term effects of exposure to air pollutants and risk of cardiovascular events. Meta-analysis is a statistical procedure of integrating results from many independent studies to attain an evidence synthesis (Egger, Smith, and Phillips, 1997). It is essentially a systematic review of numerically pooling together results of these independent studies and arriving at a summary estimate. This approach is often used by researchers to determine the effect of an observation and to what magnitude. The rationale of this research is to quantitatively examine the effects of exposure to common air pollutants and risk of hospitalizations due to acute myocardial infarction. Hence, the use of meta-analysis approach was used to synthesize effect sizes from primary epidemiological studies evaluating the short term effects of exposure to air pollutants and risk of cardiovascular events in order to obtain a summary effect. This summary effect will be compared with the air quality data to extrapolate the risk of AMI hospitalizations in Lautoka. The basic steps on the meta-analysis for this study is outline below:

3.9.1 Framing of PICO research question

The Participant-Intervention-Comparison-Outcomes (PICO) (Schardt, Adams, Owens, Keitz, & Fontelo, 2007) framework was used as a guide in developing the theory and framing of the question on air quality and risk of acute myocardial infarction hospitalizations. Information to assist in the formulation of the question was obtained from observational epidemiological studies such as case-cross over and time series studies. Hence, any Intervention was not appropriate and was replaced with the term 'Exposure'. The question was then developed using the PICO framework as a guide and is as follows:

- P - General population (both sexes, all ethnicity, all nationality)
- E - pollutants (PM2.5 and PM10)
- C - lower levels of pollutants
- O - higher levels of pollutants

On the basis of PICO, the meta-analysis research question was reframed to read, "Compared with lower levels of exposure to air pollutants, what is the risk of acute myocardial infarction related hospitalizations for people who are exposed to higher levels of air pollutants".

3.9.2 Literature Search

Based on the PICO research question, a systematic review of literature was conducted. The literature search was done to identify published primary studies that evaluated the effect of exposure to the common air pollutants and the risk of myocardial infarction. These pollutants are carbon monoxide (CO), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), ground-level ozone (O_3) and particulate matter with an aerodynamic diameter of < 2.5 microns ($PM_{2.5}$) and particulate matter with an aerodynamic diameter of < 10 microns (PM_{10}).

A comprehensive search on several electronic databases including Embase (between 1974 to December 10th, 2018) and The Ovid Medline and in-process and other non-indexed citations (between 1946 to November 27, 2018) was conducted to search for all relevant published studies. To exhaust relevant literature on the desired studies other databases were accessed which included Google scholar, Toxnet and Web of Science. In addition, the search for desired studies was extended to reference lists of eligible studies for additional published and unpublished data. In searching for relevant articles, the search terms were arranged using the Boolean logic (Lotame, 2019.). Moreover, specific search related vocabulary, symbols of truncation of expansion, and placement of the relevant terms in different sections of an article were also used to identify as much relevant studies as possible (Tuttle et. al., 2009).

The Medical Subject Headings (MeSH) classification system was employed for the primary search in Ovid Medline, Embase and Toxnet. This search included a combination of key words such as 'air pollution', 'air pollutants', 'particulate matter', 'vehicle emissions', 'ozone', 'carbon monoxide', 'sulphur dioxide', 'nitrogen dioxide' in the title or abstracts of a study. Similarly, the key words used to search for the desired outcome were myocardial Infarction, acute coronary syndrome, heart attack or heart infarction. *Particulars of the search strategy using Embase and Ovid Medline databases is attached in Appendix 1.*

3.9.3 Inclusion and Exclusion criteria

The selection of relevant studies was based on a scheme of inclusion and exclusion criteria so that only the desired studies were retrieved. The scheme included:

- All studies that was selected had to be published online from the beginning of 2008 to the year 2018.
- Studies had to be primary studies and published only in the English language.
- All qualitative studies were excluded and all duplicates were removed.
- Studies that evaluated the outcome of interest for the research i.e. outcomes had to be either 'myocardial infarction' or 'acute myocardial infarction' or -ST-Elevation Myocardial Infarction' were retained and others excluded.
- Studies had to include exposure to any of the common air pollutants which includes particulate matter with an aerodynamic diameter < 2.5 microns and < 10 microns, carbon monoxide, ground-level ozone, sulphur dioxide and nitrogen dioxide.
- All studies that focused on special population groups was excluded and those studies that addressed the general population was included.
- Moreover, primary studies which employed the case-crossover (Maclure 1991) designs were selected and studies of any other design were excluded. Case-cross over designed studies is often applied in epidemiological studies if the exposure is intermittent, the effect on risk is immediate and transient and the outcome is sudden or unexpected (Maclure and Mittleman, 2000). In addition, this study design has been used to study changes in exposure levels, progression effects on risk and outcomes with subtle but harmful effects. Moreover, in this study design cases are used and comparisons of an individual's exposure experience are made just before an event with exposure at other time periods of interest. An advantage of case-crossover design is that each case serves as its own control and confounding risk factors that are invariant and slowly changing such as gender, age, race, socio-economic status and smoking is controlled for by the design itself. Furthermore, this study design is widely used in epidemiological studies to **quantify the risk** of a population after exposure to an environmental contaminant such as air pollutants.

In case cross over studies, instead of obtaining information from two groups (cases and controls), the exposure information is obtained from the same case group but during two different periods of time. In the first period exposure is measured immediately before disease onset. In the second period exposure is measured at an earlier time (supposed to represent background exposure in the same person). Exposure among cases just prior disease onset is then compared to exposure among the same cases at an earlier time. Each case and its matched control (himself) are therefore automatically matched on many characteristics (age, sex, socio economic status, etc.) all qualitative studies were excluded and all duplicates were removed.

3.9.4 Data Extraction

The full text of the studies selected from step 3 was examined to ensure that it contained the required information. Relevant information was then extracted from all eligible studies obtained in accordance with the inclusion and exclusion criteria in the study selection process. Each eligible study was printed in PDF format and the full text read independently.

A ‘Summary of Findings’ form was created in Microsoft excel and a full description of each study characteristics was extracted as follows: the author and year of publication, the locality, the pollution exposure, the nature of the outcome, the study design, pollutant models, adjustments performed, effect measurement/effect size, the number of participants for the study. To demonstrate the strength and reliability of each study, a critique quality scale was adapted from (Mustafić et al., 2012) in order to assign a quality score to each study.

3.9.5 Constructing a Forest Plot

From the information collected in steps 4 & 5, the dataset was imported into RStudio for further analysis. The initial results were in the form of a ‘forest plot’ that graphically displayed the distribution of the effect size of the different studies in both the overall analysis and subgroup analysis.

3.9.6 Testing for heterogeneity

In this test, the information obtained in steps 4 & 5 was first used to test the heterogeneity of the studies. Evaluating the statistical heterogeneity of the studies involved using a variation of chi-square test based on pooled estimate, the effect estimate of each individual study and the number of studies. This test is referred to as Q statistic test (Cochran 1954) and the associated p-value noted, and further evaluated at 0.05 for the null hypothesis. The null hypothesis was that, "the results of the study are similar to each other or that there is no difference between the results of the studies included in the meta-analysis. The alternative hypothesis was that, "the results of the study differ from each other". If the p-value rejects the null hypothesis, then the studies are heterogeneous; if the p-value fails to reject the null hypothesis, then the conclusion will be that the studies are homogeneous. If the studies are homogeneous, the results of the studies will be pooled together and two types of estimates reported: (1) fixed effects estimates based on the assumption that the studies that have been included in the research from an exhaustive set of studies; and (2) a random effect estimate where it will be assumed that the set of studies being included in the analysis from a 'sample' or random sample of studies of 'all possible studies'. To evaluate the degree of inconsistency among studies, I^2 statistic was used to calculate the percentage of the total variation across studies was due to heterogeneity than by chance (Che et al. 2014). The scale to demonstrate the degree of heterogeneity was adopted from (Mustafic et al. 2012) where an I^2 value of >50% showed a statistically significant heterogeneity. I^2 25% or less was deemed as a low degree of heterogeneity, 25 to 75% was deemed to show a moderate degree of heterogeneity and 75% or more was deemed to have a high degree of heterogeneity.

3.9.7 Testing for Publication Bias

Here I tested for publication bias. Publication bias refers to a bias that occurs due to the fact that smaller studies and those with "equivocal estimates"(i.e. estimates that are inconclusive or those studies with negative estimates) are less likely to be published and therefore less likely to be captured in this meta-analysis than those studies that are large and have significant findings. Plotting the variance of the study estimates (variance of the effect estimate of a study is a function of its sample size) and the effects estimate itself will show that the cloud of point may define a funnel. The base of the funnel will be formed by studies that are small in size (hence large variance) and the effect estimates will vary all around the point estimate; the apex or peak of the funnel will be formed by those studies that are large sized (hence low variance) and all the estimates will be clouded around the point estimate obtained in the meta-analysis. If part of the funnel is missing, then that indicates that there was publication bias. This is referred to as the funnel plot. There are other tests, such as

'Egger's test' that can statistically report the extent of publication bias. Eggers test (Egger et al., 1997) is a statistical test to assess the funnel plot asymmetry. In addition, Egger's test plots the regression line between precision of the studies (independent variable) and the standardized effect (dependent variable). When there isn't any publication, the regression line originates in the y-axis zero. If it is much further away from zero, this suggest further evidence of publication bias (Molina, 2018). Nothing much can be done to remedy publication bias other than searching for 'fugitive literature' and contacting the research groups and other who can have studies that are small and remained unpublished or actually obtain the raw data from different sources.

3.9.8 Subgroup analyses

In the final step, 2 subgroup analysis was performed. Here, I sub grouped the data and analyzed them separately using a regression model. I tested if the overall estimates was different for those in WHO Western Pacific Region countries and also tested whether the overall estimates was different for those studies that examined the risk of AMI hospitalization associated with at least each 5-10$\mu g/m^3$ increases in $PM_{2.5}$

3.9.9 Quality score assessment

The quality score used to assess the methodological quality of the selected studies were adapted from a scale based on previous systematic reviews and meta-analysis. The four components used to evaluate these articles are as follows: (1) Determining of MI (0 to 1 point). A score of 1 was given if the diagnosis of MI was coded in accordance with the International Classification of Diseases or based on angiographic criteria or in accordance with the clinical, laboratory and electrocardiographic criteria. A score of 0 was given if there was no description of diagnosis or the diagnosis was determined by patient history or other criteria. (2) The quality of air pollutant measurements (0 to 1 point). If measurements were performed at least daily and less than 25% missing data, a score of 1 was given. Measurements that were not performed hourly and more than 25% missing data or there was no description of particulate measurements, a score of 0 was given. (3) Adjustment for confounders (0 to 1 point). Due to the differences in research methods used between case-crossover and time series studies, the methods of adjustment for confounders were also different. For time series studies, a score of 1 was given if adjustment for covariates was performed for several important covariates together, including long-term trends, seasonality, temperature, humidity, pressure or day of week. For case-crossover studies which control invariant and slowly changing confounders by the design itself, therefore, if an adjustment was made for temperature, humidity, pressure or day of week, a score of 1 was given. A score of 0 was given if studies did not do any adjustment of the above mentioned important covariates. Finally, if a study got maximum score in each component, it was considered to be of good quality. On the other hand, if one of the 3 components got a minimum score of 0, it was considered to be of low quality.

CHAPTER 4

RESULTS

4.1 Summary

This chapter highlights the output obtained from the methodologies used. The mean concentrations of the pollutant PM_{10} during this study was observed to be significant. The mean concentrations of PM_{10} in the duration of the study and the imputed daily mean concentrations both exceeded the WHO recommended guideline value. Even though the mean concentrations of $PM_{2.5}$ were relatively low. Its presence even in low concentrations as observed in this study is still of great health concern, in particular on the objectives of this study. Concentrations of other pollutants were insignificant but their presence is worth noting and is deliberated further in the next chapter. The concentrations of both PM_{10} and $PM_{2.5}$ during the hours of a day suggests that contributions from vehicles is quite significant. The systematic review and meta-analysis provided some evidence in both the overall and subgroup analyses of the relationship between short-term exposure to air pollutants and risk of AMI hospitalizations. There was evidence of high heterogeneity and low heterogeneity in both the overall and subgroup analyses respectively. Publication bias was present in both overall and subgroup analyses and some factors that may contribute to this biases is discussed in the next chapter.

4.2 Introduction

The objective of this chapter is to firstly, provide descriptive information on the ambient air pollutant levels in the city of Lautoka will be presented. Air pollutants that showed significant levels will be visualized using graphs to highlight existing variations with time. The mean and range will be imputed to show the mean daily pollutant levels in comparison to the WHO air pollutant guideline values. Correlation coefficients will also be imputed and presented to demonstrate the statistical relationship between two variables. Secondly, the forest plot and the precise pooled estimate derived from the meta-analysis on the risk of myocardial infarction due to exposure to air pollutants will also be presented. Lastly, on the basis of the pooled estimate from the meta-analysis and air quality data the risk of myocardial infarction hospitalization will be predicted.

4.3 Descriptive statistics on air pollutants and weather variables

4.3.1 Summary statistics of the exposure variables

Table 4: Summary of the distribution of ambient air pollutant levels and meteorology variables in the Lautoka CBD

		Percentiles				
Exposure Variables	**Mean± SD**	**Min**	**25%**	**50%**	**75%**	**Max**
PM_{10} (µg/m^3)	73.42 ± 33.23	30.71	44.88	68.97	99.57	142.76
$PM_{2.5}$ (µg/m^3)	10.87 ± 5.8	3.95	8.14	9.44	12.82	31.75
SO_2 (ppb)	0.09 ± 0.17	0.01	0.03	0.03	0.06	0.76
NO_2 (ppb)	0.05 ± 0.03	0.02	0.03	0.04	0.04	0.17
CO (ppb)	0.39 ± 0.22	-0.41	0.2	0.32	0.42	0.63
O_3 (ppb)	0.0 ± 0.03	-0.0	0.0	0.0	0.01	0.11
Temp(°C)	27.53 ± 2.07	23.32	26.72	27.53	29.21	30.56
Rel. Humidity (%)	68.72 ± 6.77	59.4	63.7	67.36	71.94	83.3
Pressure (hPa)	1012 ± 2.95	1004	1011	103	1014	1015

Table 4 provides the distribution of ambient air pollutant levels and meteorological variables during the study period. The overall mean daily PM_{10} and $PM_{2.5}$ concentrations and standard deviation was 73.42 ± 33.23 g/m^3 and 10.87 ± 5.8 µg/m^3 respectively. The range for PM_{10} concentrations was a minimum concentration of 30.71 to a maximum concentration of 142.76 µg/m^3 whereas the range for $PM_{2.5}$ concentrations was a minimum concentration of 3.95 to a maximum concentration of 31.75 µg/m^3. The overall mean concentrations and standard deviation for the pollutants SO_2, NO_2, O_3 and CO were 0.09 ± 0.17 ppb, 0.05± 0.03 ppb, 0.004 ± 0.03 ppb and 0.39 ± 0.22 ppb respectively. The range for the SO_2 concentrations was a minimum concentration of 0.012 to a maximum concentration of 0.76ppb, the range for NO_2 was a minimum concentration of 0.02 to a maximum concentration of 0.173 ppb, range for CO was a minimum concentration of -0.41 to a maximum concentration of 0.63ppb, range for ozone was a minimum concentration of -0.0009ppb to a maximum concentration of 0.109 ppb. The mean daily temperature was 27.53 ± 2.07 °C with a range of a minimum temperature of 23.32 to a maximum temperature of 30.56 °C. The mean relative humidity was 67.72 ± 6.77% with a range of a minimum relative humidity of 59.4 to a maximum relative humidity of 83.3 %. The mean atmospheric pressure was 1012 ± 2.95 hPa with a range of minimum atmospheric pressure of 1004 hPa to a maximum atmospheric pressure of 1015 hPa.

4.3.2 Daily averages of the air pollutants and weather variables

Table 5: Mean daily levels of air pollutants and weather variables during the days of the week

Days	$PM_{2.5}$	PM_{10}	O_3	CO	SO_2	NO_2	Pressure	Humidity	Temperature
Monday	7.22	40.77	0.0	-1.88	0.01	0.01	1014.3	60.31	27.4
Tuesday	8.62	62.28	0.0	0.4	0.03	0.02	1013.3	74.58	27.63
Wednesday	8.89	59.83	0.01	0.27	0.2	0.03	1013.68	66.02	27.33
Thursday	12.65	77.92	0.02	0.3	0.04	0.06	1012.26	68.23	27.25
Friday	12.98	93.28	0.03	0.38	0.06	0.04	1012.19	65.49	28.21
Saturday	5.71	50.85	0.02	-0.1	-0.0	0.1	998.97	58.0	30.27

Table 5 illustrates the mean levels of air pollutants and weather variables in a typical week (Monday to Saturday) during this study. Note there are no mean concentrations for Sunday as this day was dedicated for data extraction from the data logger as well as checking for the power in the batteries. The concentrations for almost all pollutants indicated very low concentrations at the beginning of the week, Mondays, and it increased towards the Fridays. The air pollutant levels showed a marked decreased on Saturday. The weather variables did not show a specific pattern as compared to the air pollutants.

4.3.3 Averages for air pollutants and weather variables for each hour of a day for 24hrs

Table 6: Mean levels of the air pollutants and weather variables in the hours of the day

Hours	PM_{10}	$PM_{2.5}$	NO_2	SO_2	O_3	CO	Temp.	Humidity	Pressure
1	77.23	8.16	0.03	0.03	0.0	0.31	27.0	69.41	1010.57
2	36.55	6.02	0.04	0.03	0.01	0.18	26.66	71.43	1003.72
3	30.71	4.59	0.17	0.04	0.0	-0.41	25.29	75.9	1007.86
4	35.19	3.94	0.03	0.05	-0.0	0.2	23.71	81.33	1013.06
5	39.82	4.18	0.03	0.06	-0.0	0.23	23.32	83.3	1013.36
6	130.15	9.6	0.04	0.07	-0.0	0.38	23.85	80.9	1013.87
7	94.4	12.66	0.05	0.06	-0.0	0.63	25.84	73.71	1014.49
8	126.59	31.75	0.04	0.05	0.01	0.6	27.81	66.84	1015.07
9	106.42	15.25	0.03	0.01	0.01	0.32	29.17	63.67	1015.15
10	100.11	10.39	0.06	0.76	0.02	0.21	30.13	59.4	1015.01
11	41.43	8.29	0.07	0.46	0.11	0.01	30.56	59.82	1014.41
12	47.78	8.1	0.03	0.2	0.1	0.16	29.89	61.65	1013.47
13	45.55	8.3	0.02	0.03	0.01	0.43	30.0	61.17	1013.55
14	42.88	8.15	0.02	0.01	0.01	0.39	29.72	61.75	1013.01
15	47.47	9.29	0.03	0.02	0.0	0.39	29.33	63.95	1012.59
16	60.72	10.25	0.03	0.02	0.0	0.45	29.1	63.71	1012.54
17	78.0	11.65	0.13	0.03	0.05	-0.08	28.75	65.54	1012.77
18	102.17	13.28	0.04	0.15	0.03	0.47	27.89	66.77	1013.03
19	99.39	15.88	0.03	0.03	0.0	0.41	27.43	66.64	1010.49
20	142.76	18.89	0.04	0.03	0.0	0.46	26.95	71.29	1011.01
21	85.07	14.62	0.04	0.03	0.0	0.34	26.74	71.53	1012.07
22	79.44	10.57	0.04	0.03	0.0	0.28	26.79	73.16	1015.07
23	57.73	9.09	0.04	0.03	0.01	0.26	27.64	68.43	1005.4
24	54.55	7.96	0.03	0.03	0.0	0.33	27.16	67.87	1010.59

Table 6 illustrates the mean levels of air pollutants and weather variables in a typical week (Monday to Saturday) during this study. Note there are no mean concentrations for Sunday as this day was dedicated for data extraction from the data logger as well as checking for the power in the batteries.

4.4 Correlation Coefficients

Table 7: Pearson's correlation imputed showing the relationships between the air pollutants and weather variables

Variables	PM_{10}	$PM_{2.5}$	SO_2	NO_2	CO	O_3	Temp.	Humidity	Pressure
PM_{10}	1	0.76	0.05	-0.14	0.51	-0.2	-0.05	0.02	0.33
$PM_{2.5}$		1	-0.07	-0.14	0.52	-0.09	0.19	0.22	0.3
SO_2			1	0.1	-0.21	0.48	0.38	-0.4	0.3
NO_2				1	-0.78	0.18	-0.1	0.11	-0.18
CO					1	-0.38	0.05	-0.09	0.31
O_3						1	0.53	-0.49	0.2
Temp.							1	-0.98	0.2
Humidity								1	-0.17
Pressure									1

Table 7 displays the Pearson's correlation coefficients, r, and the relationships between the air pollutants and weather variables. A strong (positive) linear relationship was observed between PM_{10} and $PM_{2.5}$ with a correlation coefficient, r = 0.76, p-value<0.001. A moderate (positive) linear relationship was observed between PM_{10} and carbon monoxide, r = 0.51 and p=0.001. This moderate (positive) linear relationship was also exhibited between PM10 and atmospheric pressure with a correlation coefficient, r = 0.33, p=0.2. No linear relationship was observed between PM_{10} and SO_2, NO_2, O_3, temperature or relative humidity. PM2.5 showed a moderate (positive) linear relationship with CO, r = 0.52, p=0.008. A weak (positive) relationship was observed between $PM_{2.5}$ and atmospheric pressure, r = 0.3, p = 0.16. No linear relationship was observed between $PM_{2.5}$ and SO_2, NO_2, O_3, temperature or relative humidity. SO_2 showed a moderate (positive) linear relationship with the variables O_3 (r = 0.48, p=0.02), temperature (r = 0.38, p = 0.06) and pressure (r = 0.3, p =0.15). SO_2 also exhibited a moderate (negative) linear relationship with the weather variable, humidity (r = -0.4, p=0.06). No linear relationship was observed between SO_2 and NO_2 or CO. A strong (negative) linear relationship was observed between NO_2 and CO (r = -0.78, p<0.001). No linear relationship was observed between NO_2 and O_3, temperature, relative humidity or atmospheric pressure. CO showed a weak (positive) linear relationship and a weak (negative) linear relationship atmospheric pressure (r = 0.31, p = 0.14) and O_3 (r = -0.38, p = 0.06) respectively. No linear relationship was observed between CO and the weather variables temperature or relative humidity. A moderate (positive) relationship was observed between O_3 and the weather variable temperature, r = 0.53, p = 0.007. A weak (negative) linear relationship was also observed between O_3 and relative humidity, r = -0.49, p=0.01. No linear relationship was observed between O_3 and atmospheric pressure. Temperature showed a strong (negative) linear relationship with relative humidity (r = -0.98, p <0.001). There was no linear relationship observed between temperature and atmospheric pressure. Moreover, no linear relationship was observed between relative humidity and atmospheric pressure.

4.5 Histograms for the weather variables

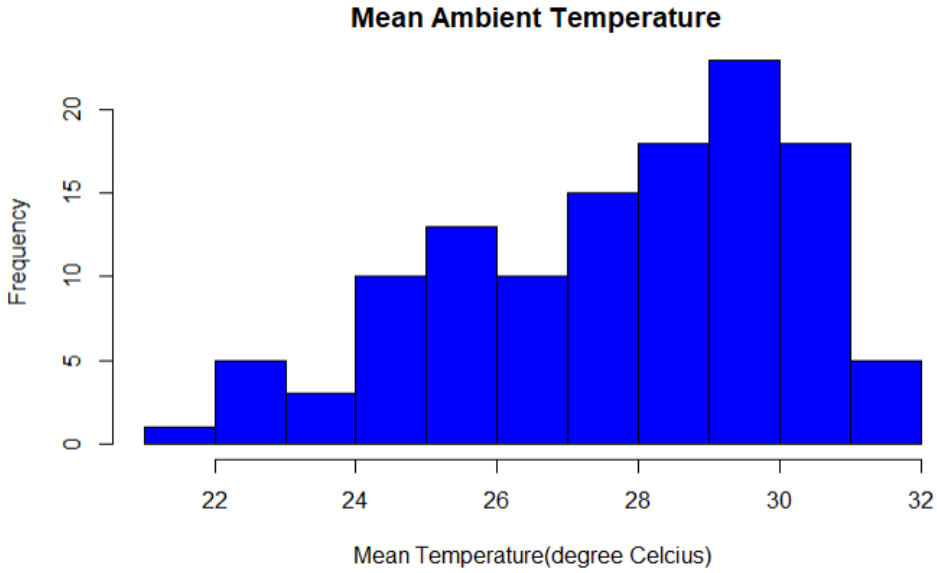

Figure 15: Histogram for the mean temperature for the duration of the study in degrees Celcius

Figure 15 illustrates the frequency distribution of the mean temperature (expressed as degrees Celsius, °C) for the Lautoka CBD. On the horizontal axis or x-axis is the mean temperature values and on the vertical axis or y-axis is the frequencies of these temperature values. Most of the days recorded a mean temperature between 27°C/28°C and 29°C/30°C whilst very few days recorded a temperature of less than 25°C. The range was from 23.32°C to 30.56°C.

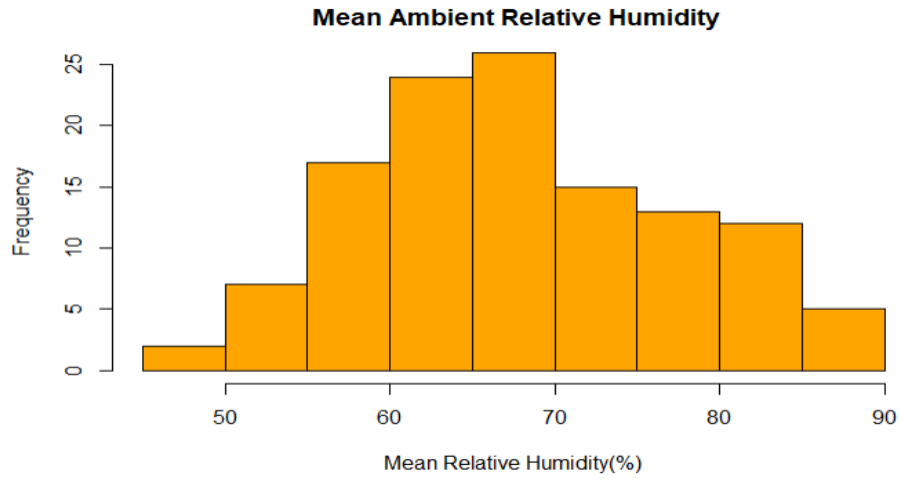

Figure 16: Histogram for the mean relative humidity for the duration of the study in percentage

Figure 16 illustrates the frequency distribution of the mean relative humidity (expressed as percentage, %) for the Lautoka Central Business District. The horizontal axis or x-axis is the mean relative humidity values and on the vertical axis or y-axis is the frequencies of these rel. humidity values. Most of the days recorded a mean rel. humidity of 60 % to 70% whilst very few days had a rel. humidity of 75% to 80%. The range was from 59.4% - 83.3%.

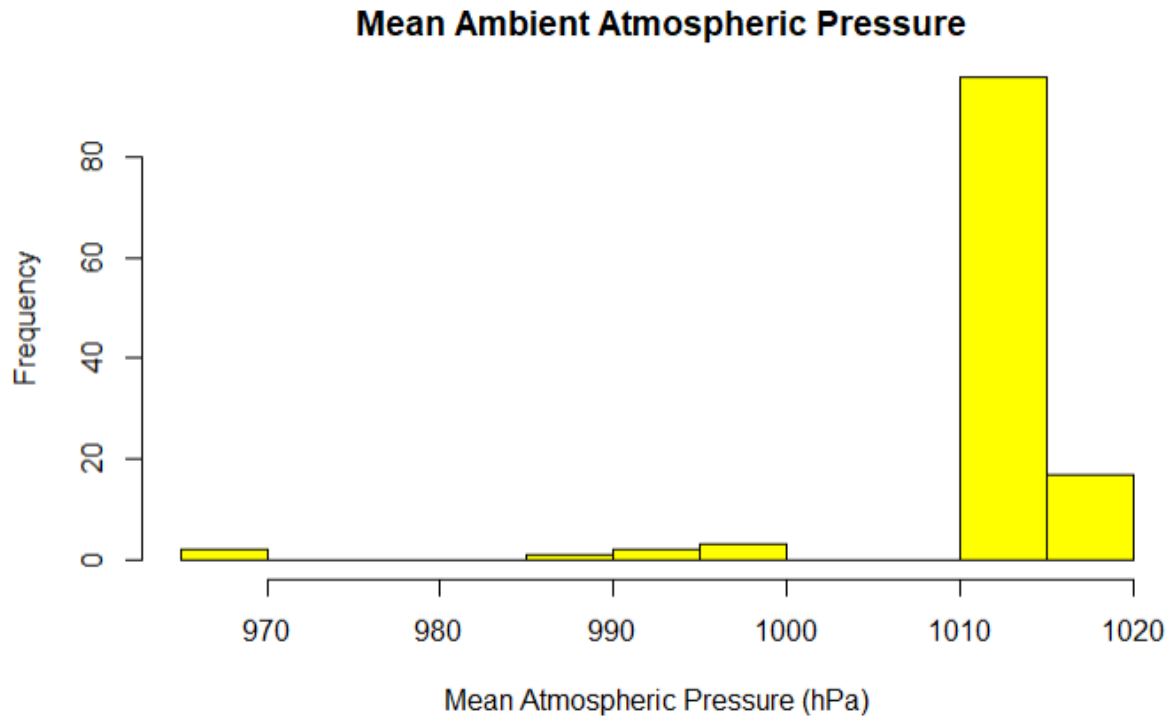

Figure 17: Histogram for the mean atmospheric pressure for the duration of the study in hPa

Figure 17 illustrates the frequency distribution of the mean atmospheric pressure (expressed as hectopascal, hPa) for Lautoka city. The horizontal axis or x-axis, is the mean atmospheric pressure in hPa and on the vertical axis or y-axis is the frequencies of the atmospheric pressure values. Most of the days recorded a mean atmospheric pressure between 1010 to 1015 hPa. The range was from 1004 to 1015hPa

4.6 Time series Graphs

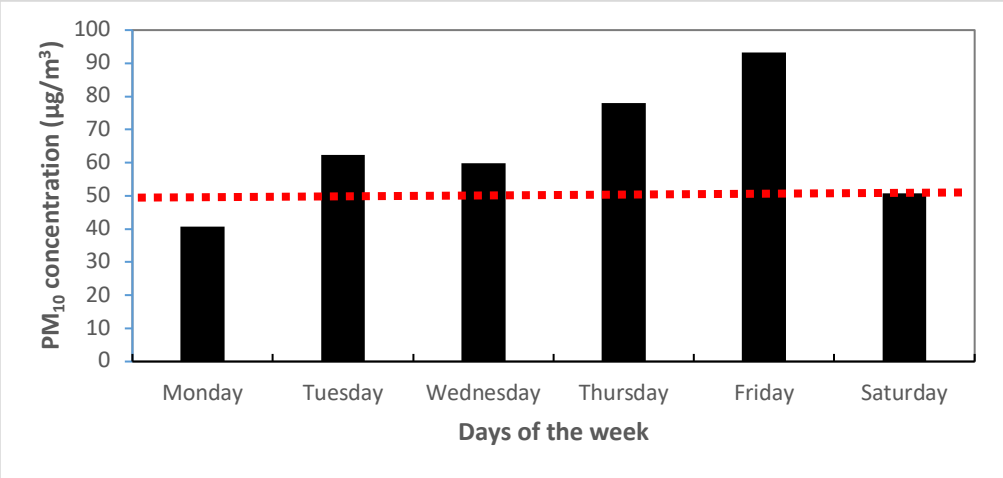

Figure 18: Daily mean PM_{10} concentrations for each day of the week

The bar graph illustrates the mean PM_{10} concentrations for each day of the week (Mon-Sat) for the Lautoka CBD with the horizontal axis showing the days of the week and the vertical axis showing the mean PM_{10} concentrations in micrograms per cubic meter ($\mu g/m^3$). The highest daily mean concentrations was recorded for Fridays whilst the lowest concentrations were on the Mondays. The daily mean concentrations was 64.15 $\mu g/m^3$ with a range of 40-77$\mu g/m^3$ -93.28$\mu g/m^3$. Insufficient data was collected on Sundays as the batteries were recharged and data extracted on this day in a week. The dotted red line indicates the WHO 24 hour average guideline value of 50$\mu g/m^3$. The dotted blue line indicates the trend at which the mean concentrations are being observed from Monday to Saturday. The highest concentrations were recorded on Friday and the least was recorded on Monday.

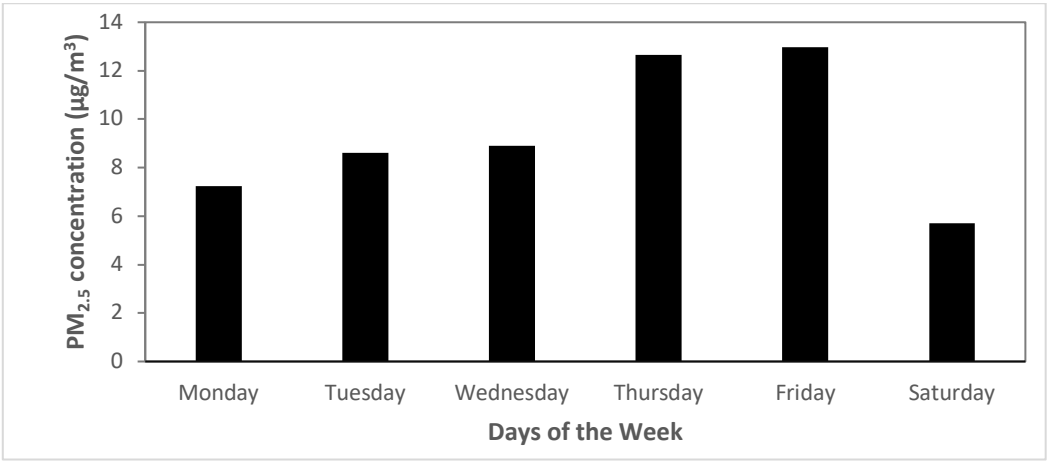

Figure 19: Daily mean $PM_{2.5}$ concentrations for each day of the week

The bar graph illustrates the mean $PM_{2.5}$ concentrations for each day of the week (Mon-Sat) for the Lautoka CBD with the horizontal axis showing the days of the week and the vertical axis showing the mean $PM_{2.5}$ concentrations in micrograms per cubic meter (($\mu g/m^3$). The daily mean concentrations was 9.34 $\mu g/m^3$ with a range of 5.7$\mu g/m^3$ – 12.98$\mu g/m^3$The dotted blue line indicates the trend at which the mean concentrations are being observed from Monday to Saturday. The highest concentrations were recorded on Friday and the least was recorded on Saturday.

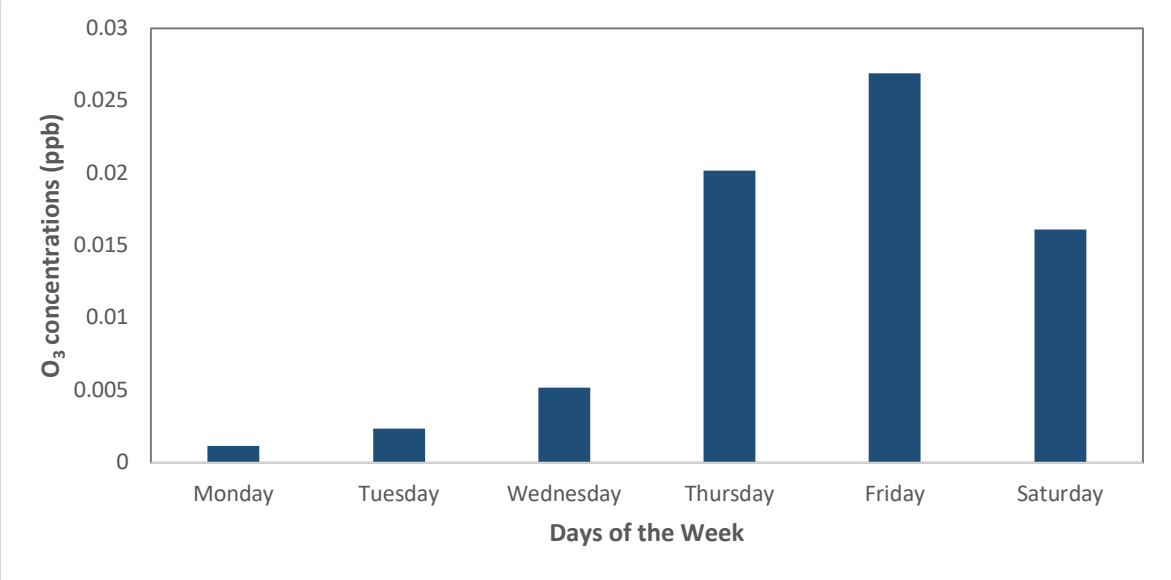

Figure 20: Daily mean O_3 levels for each day of the week

The above bar graph illustrates the mean O_3 concentrations for each day of the week (Mon-Sat) for the Lautoka CBD with the horizontal axis showing the days of the week and the vertical axis showing the mean PM2.5 concentrations in micrograms per cubic meter ((µg/m3). The dotted blue line indicates the trend at which the mean concentrations are being observed from Monday to Saturday. The highest concentrations were recorded on Friday and the least was recorded on Saturday.

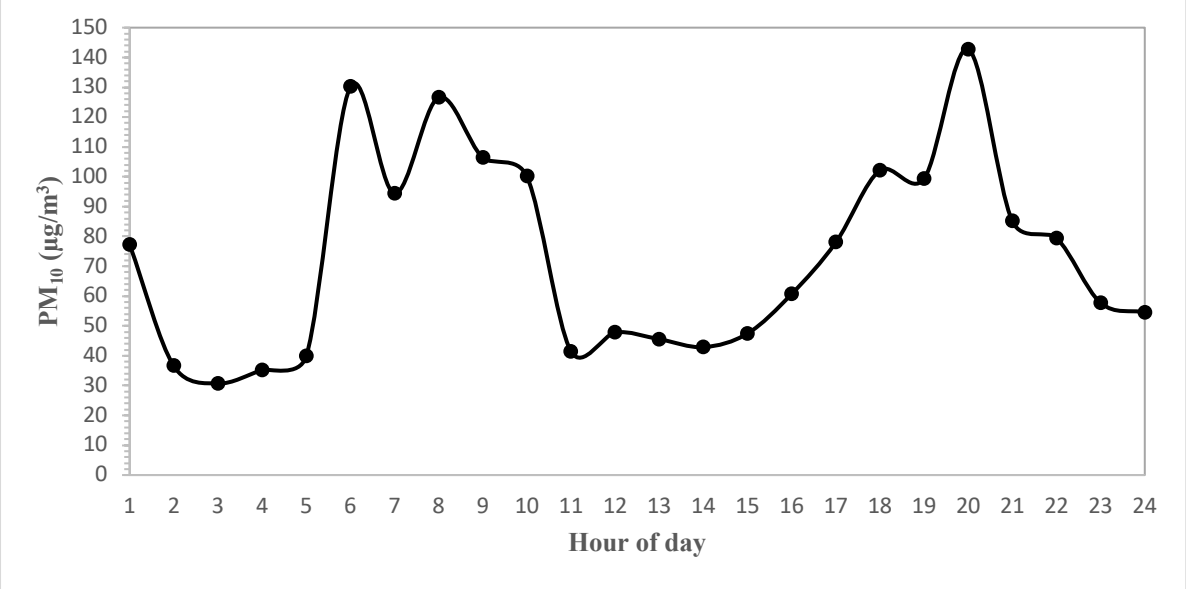

Figure 21: Mean levels of ambient PM_{10} for the hours of a day

Figure 21 shows the hourly mean concentrations of ambient PM_{10} observed for the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the pollutant PM_{10} in µg/m^3. From this graph, it is obvious that ambient PM_{10} levels increased significantly early in the morning between 5am and 9am. It then decreased towards mid-day and then increased again 3pm and reaching its highest levels (142.76µg/m^3) at 8pm in the evening before decreasing. The early morning hours between 2am to 5am recorded the lowest (30.7µg/m^3) ambient PM_{10} levels.

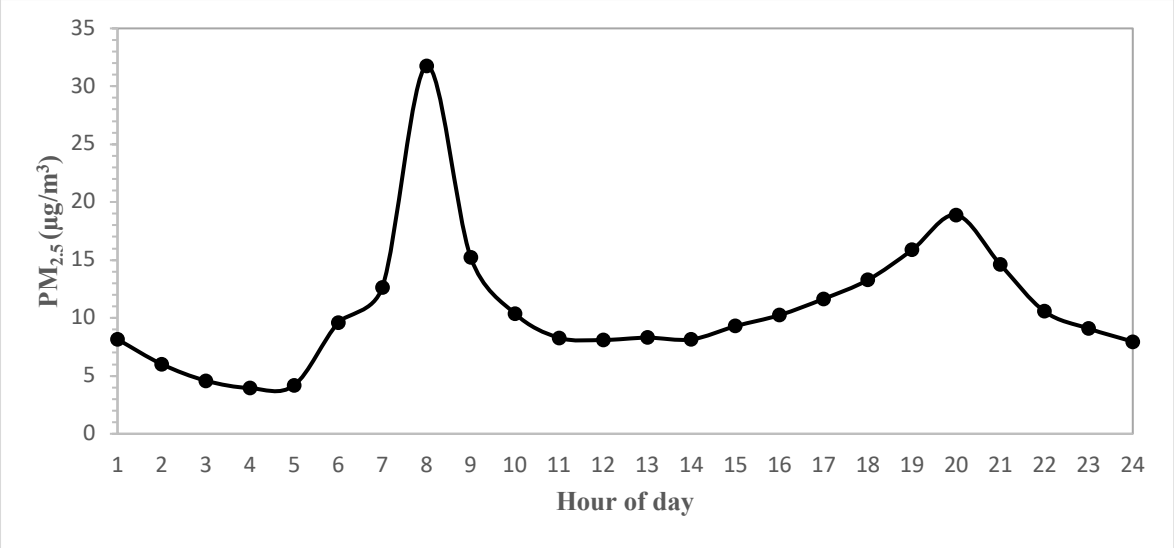

Figure 22: Mean levels of ambient $PM_{2.5}$ for the hours of a day

Figure 22 shows the hourly mean concentrations for ambient $PM_{2.5}$ observed during the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the pollutant $PM_{2.5}$ levels in µg/m^3. From this graph, it can be deduced that $PM_{2.5}$ levels increased significantly between 5 am to 8am in the morning. The highest levels of ambient $PM_{2.5}$ was observed at 8am. The levels decreased after 8am and slightly increased again from 3pm in the afternoon to 8pm in the evening.

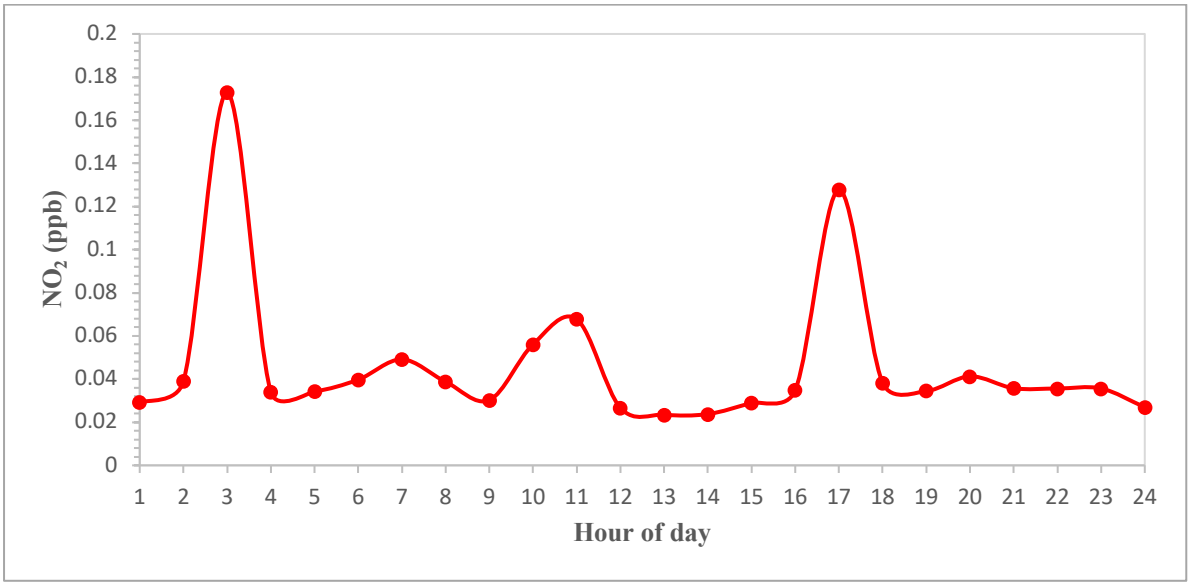

Figure 23: Mean levels of ambient NO_2 for the hours of a day

Figure 23 shows the hourly mean concentrations for ambient NO_2 observed for the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the pollutant NO_2 in µg/m^3. From the graph, it appears that NO_2 levels was at its highest (0.173ppb) at 3am in the morning. It then decreased until 4am, fluctuated between 5am and 3pm and then had a slight increase from 4pm to 5pm. It decreased again until 6pm and fluctuated towards midnight. The lowest ambient NO_2 levels was recorded at 1pm (0.0234 ppb).

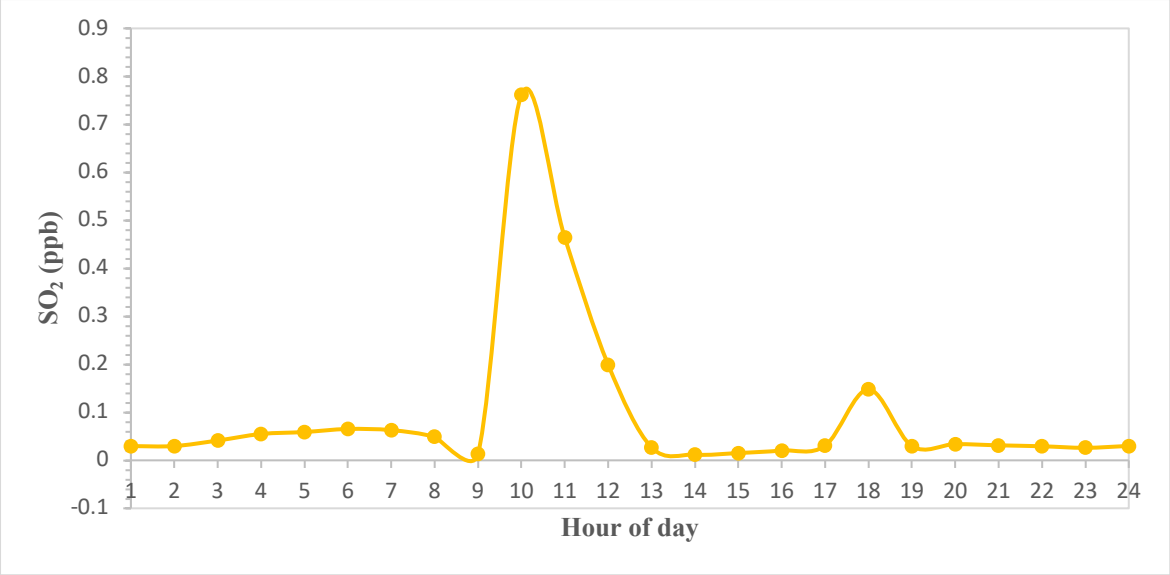

Figure 24: Mean ambient SO_2 levels for the hours of a day

Figure 24: shows the hourly mean concentrations for ambient SO_2 observed for the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the pollutant SO_2 levels in µg/m^3. Ambient SO_2 levels was at its highest (0.76ppb) at 10am in the morning. The lowest SO_2 level (0.012ppb) was recorded at 9am. From1pm it evened off towards midnight with an exception of a slight increase between 5pm and 7pm.

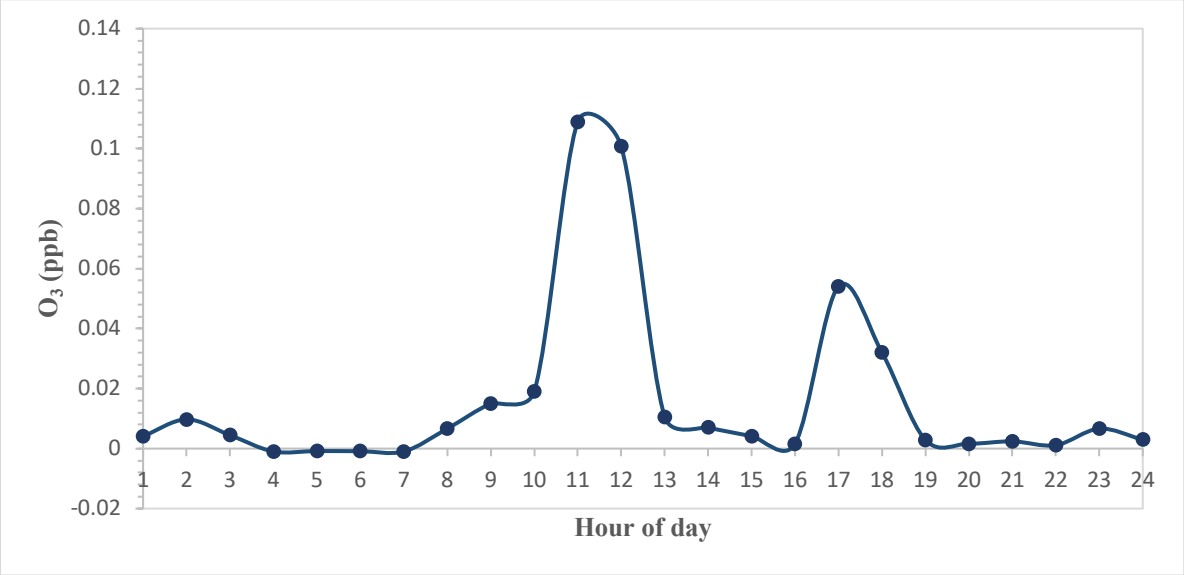

Figure 25: Mean ambient O_3 levels for the hours of a day

Figure 25 shows the hourly mean concentrations for ambient O_3 observed for the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the pollutant O_3 levels in µg/m^3. The graph illustrates that ambient O_3 levels was at highest (0.109ppb) between 10am in the morning and 1pm in the afternoon. Another slight increase was observed between 4pm and 7pm in the afternoon. From 7 pm to 7am, ambient O_3 levels were at very low levels with the lowest (-0.0009 ppb) recorded at 7am.

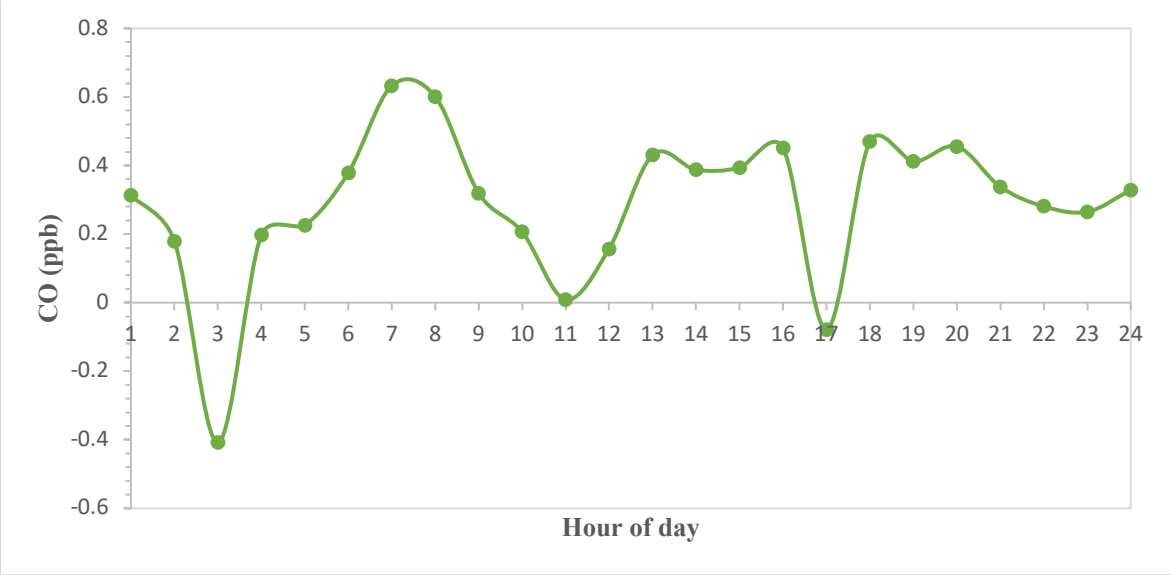

Figure 26: Mean levels of ambient CO for the hours of a day

Figure 26 shows the hourly mean concentrations for ambient CO observed for the study duration in Lautoka with horizontal axis representing the time (hours of a day) and the vertical axis representing the pollutant CO levels in ppb(parts per billion). From the graph, there was a lot of fluctuations in the ambient CO levels. Ambient CO levels increased between 4am and 7am. It then decreased until 11am and increased again until 1pm. CO levels was almost constant between 1pm to 4pm before decreasing until 5pm. It increased again after 5pm and the levels was almost constant into the midnight to early morning. The highest ambient CO levels (0.63ppb) was recorded at 7am whilst the lowest ambient CO level (-0.41ppb) was recorded at 3am.

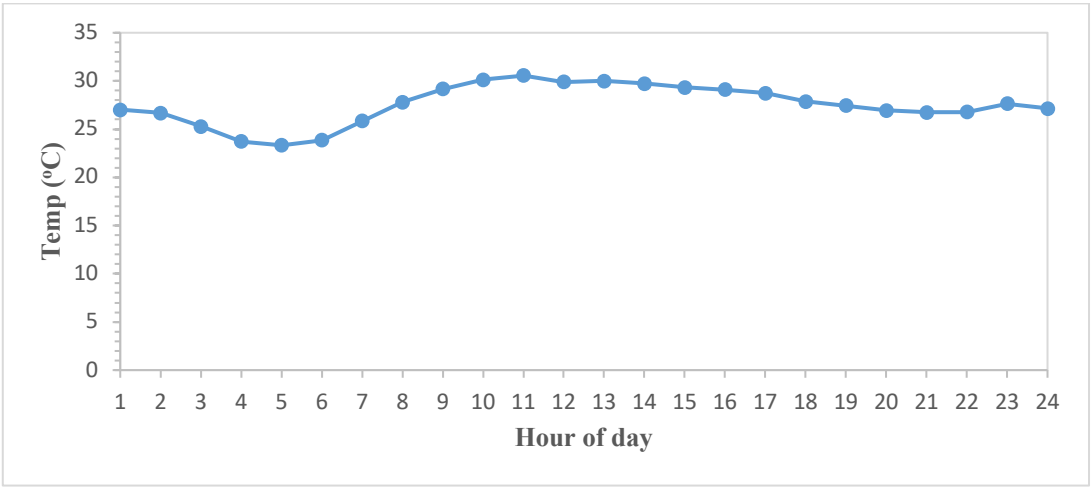

Figure 27: Mean ambient temperature levels for the hours of a day

Figure 27 shows the hourly mean concentrations for the variable Temperature for the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the weather variable Temperature in degrees Celsius (°C).The graph shows that higher temperatures were recorded between 9am in the morning and 4pm in the afternoon. As expected, the temperature started to drop in the early hours of the morning commencing after 1am until 5am. After 5am the temperatures started to increase again. The highest mean temperature recorded was 30.56°C and the lowest temperature was 23.32°C.

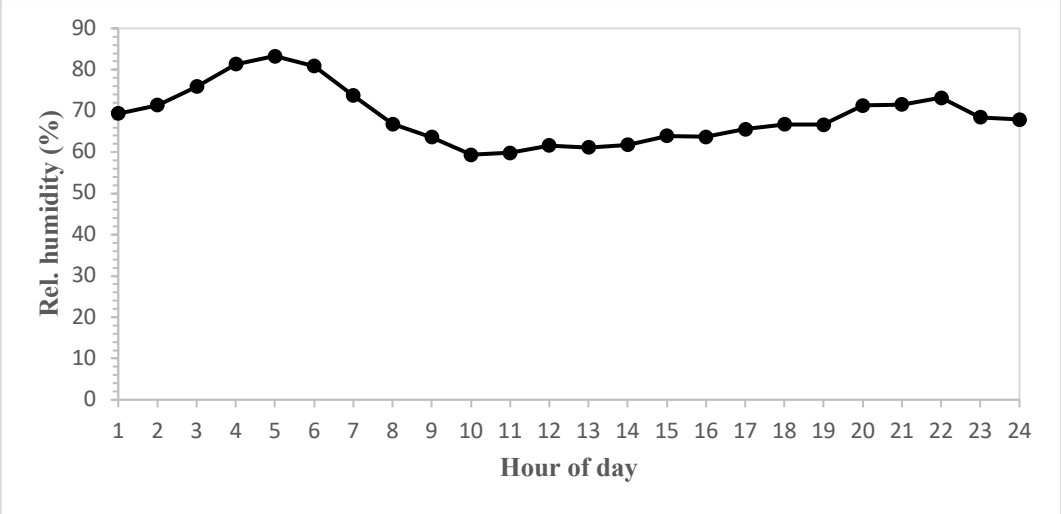

Figure 28: Mean ambient relative humidity for the hours of a day

Figure 28 shows the hourly mean concentrations for the variable Relative Humidity for the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the weather variable, Relative humidity in percentage (%). The graph shows that relative humidity levels was high in the early hour of the morning between 3am to 6am with the highest levels recorded at 5am. After 6am the relative humidity levels dropped until 10am where it slightly increased towards late in the evening. The highest relative humidity levels recorded was 83.3% and the lowest was 59.4%.

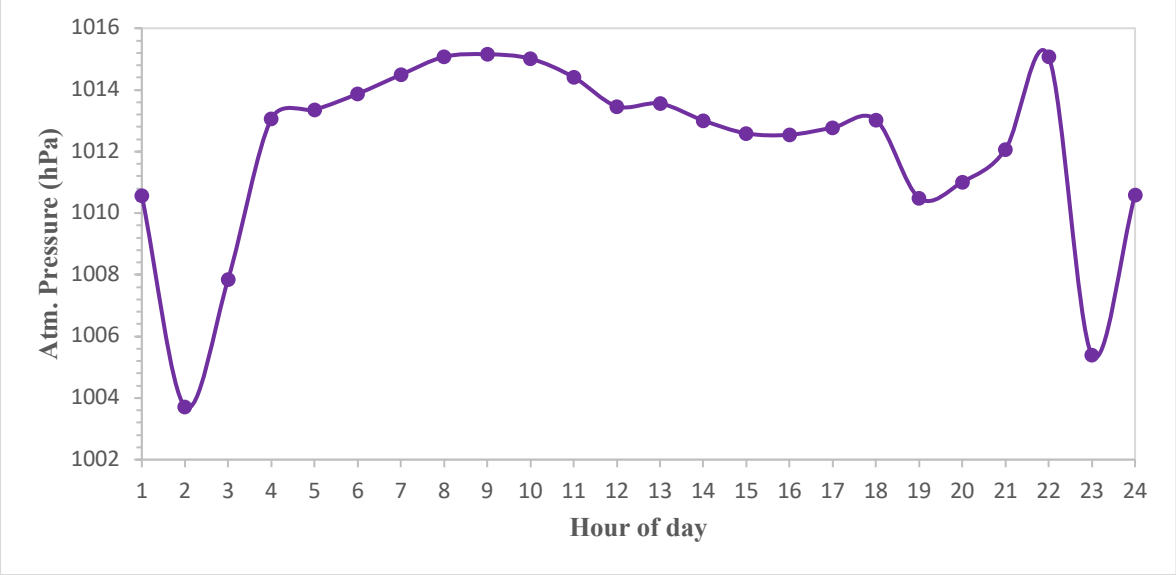

Figure 29: Mean atmospheric pressure for the hours of a day

Figure 29 shows the hourly mean concentrations of the variable atmospheric Pressure for the study duration in Lautoka with the horizontal axis representing the time (hours of a day) and the vertical axis representing the weather variable, atmospheric Pressure in hector-pascal (hPa). The graph illustrates that atmospheric pressure tend to decrease significantly on two time intervals. The first decrease was between 1am and 4am in the morning. The second decrease in atmospheric pressure was from 10pm to 11pm and increased again towards midnight. Atmospheric pressure levels during daylight (6am to 5pm) was always high and ranged between 1012 and 103 hPa. The highest atmospheric Pressure level recorded was 1015 hPa and the lowest was 1012 hPa

4.7 Description of studies included in the meta-analysis

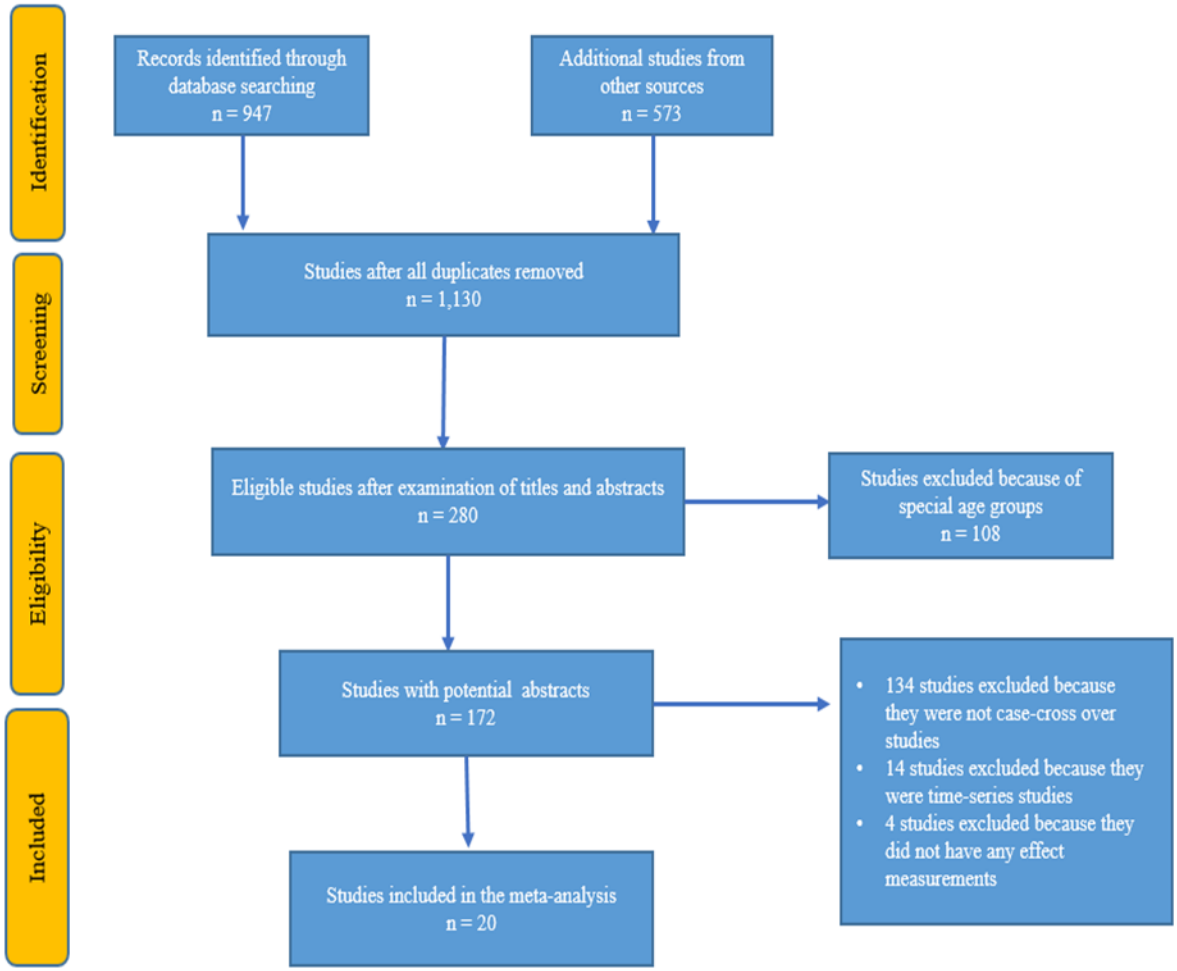

Figure 30:Flow diagram of all the included studies for the meta-analysis

The systematic search initially identified a total of 1,520 relevant studies. These studies included those that were identified through database search and those from other sources. These studies were also published in the English language, studies for humans only and published between the years 2008 and 2018 only. After reading and comparing the titles of each study all duplicates were removed and was left with 1,130 studies. By carefully reading through all titles, a further 850 studies was excluded: these were studies that did not examined the air pollutants as the exposure variable or the specific outcome variable. A total of 280 potential studies was available after which all their abstracts were carefully examined. A further 108 studies was excluded as they focus ed on special age groups and a further 130 studies were removed because they were not case-crossover studies. 42 studies were then available and 8 more were removed as they did not present any effect size measurements. 34 articles was then reviewed by an independent reviewer and a further 14 articles were excluded because of conflicting outcomes. 20 case crossover studies was then included in the meta-analysis and their description are as follows:

The study by (Chiu, Weng, Chiu, & Yang, 2017) investigated the short-term effects of ambient O_3 on the daily hospitalizations of MI. The study was conducted in Taipei, Taiwan for all MI (ICD9) patients registered under the Bureau of National Health Insurance over a 5

year period from 2006 to 2010. A time-stratified approach was used in this case-crossover study with the stratification of time into separate months to identify referent days falling on the same day of the week within the same month as the index day. A cumulative lag period of up to 2 previous days (i.e. average air pollutant concentrations of the same and previous 2 days). Conditional logistic regression was used to explore the association between the ambient concentrations of O_3 and the number of MI hospitalizations. Estimates of the associations was expressed as odds ratios and 95% confidence intervals with weights equal to the number of hospitalizations on that day. Adjustments was also made for ambient temperature, relative humidity and barometric pressure. Chiu and colleagues observed that in single-pollutant models, the increase in MI admissions was significantly associated with O_3 on both cold (<23^{o}C) and warm (>23^{o}C) days and an increase in MI admissions respectively. In 2 pollutant models MI admissions increased significantly on cool days. From these findings, Chiu and others suggested that short-term exposure to O_3 concentrations can increase the MI hospital admissions on both warm and cool days. The study by (Evans et al., 2016) examined the onset of STEMI after exposure to wood smoke and traffic pollution. This study was conducted in Monroe County, New York and included a total of 362 STEMI patients from 1st January 2007 to 30 September 2012. A time-stratified approach was employed that was similar to other case-crossover studies. Conditional logistic regression was employed to determine the relative odds of STEMI with interquartile increases in pollutant concentrations (lag0-72hr means). Adjustments were made for seasons (colder and warmer months), temperature and relative humidity. Evans and colleagues observed that for each 7.1μg/m^3 increases in $PM_{2.5}$ increased the odds of STEMI hospitalizations. In addition, the authors also observed that increases in ozone and carbon monoxide concentrations in the previous hour also increased the odds of STEMIs. From these findings, Evans and others suggests the increased levels of $PM_{2.5}$ increases the odds of STEMI. (Xiaodong et al., 2015) assessed the variations in acute myocardial infarction (AMI) hospital admissions relative to air pollutant levels between 1st November 2013 and 27 April 2014 in Shanghai, China. The pollutants included in the study are $PM_{2.5,}$ PM_{10}, NO_2, SO2, and CO whilst the definition of AMI admissions were consistent with the International Classification of Diseases, 9th revision (ICD-9). After imputing the 24 hour means for each pollutant, conditional logistic regression was used to calculate the exposure effect and the effects estimate expressed as Odds Ratio (OR) per 50μg/m^3 of each pollutant with pollutant with weights equal to the number of hospitalizations on that day. Xiaodong and colleagues observed that urban background levels of PM_{10}, $PM_{2.5}$ and CO were significantly associated with an increased risk in AMI and concluded that an increased risk in AMI is associated with short-term exposure to moderate – serious pollutant levels. The study by (Weichenthal et al., 2017) examined if hospital admissions for myocardial infarction (MI) was associated with short-term changes in outdoor $PM_{2.5}$. The study was conducted in 3 regions in British Columbia, Canada between 2008 and 2015 with a total of 2,881 myocardial infarction cases. Age and sex of participants were stratified to determine the relationship between $PM_{2.5}$ and MI. Conditional logistic regression was employed to describe the relationship between the $PM_{2.5}$ concentrations and the risk of MI. Adjustments were made for mean ambient temperature (lag0-3day). Weichenthal and colleagues observed that each 5μg/m^3 of 3-day average $PM_{2.5}$ was associated with an increased risk of MI; the authors concluded that among elderly patients a short-term change in outdoor $PM_{2.5}$ concentrations increases the risk of MI. The case-crossover study by (Sahlén et al., 2019) examined the association of short-term risk of STEMI after exposure to of air pollutants (NO_2, SO_2, O_3, $PM_{2.5}$, PM_{10}). The study was conducted in Stockholm, Sweden from January 2000 - June 2014 with a total of 14,601 STEMI patients who are registered in

SWEDEHEART (Swedish Web System for Enhancement and Development of Evidence Based Care in Heart Disease Evaluated According to Recommended Therapies). After imputing the averages of pollutant concentrations 2 weeks before and after exposure, conditional logistic regression was employed to analyze (lag0-24hrs) before hospital admissions. Multivariate single-pollutant models was also built with adjustments made for atmospheric pressure, precipitation relative humidity, wind velocity and temperature. Sahlen and colleagues observed that the risk of STEMI with NO_2 was strongest at 15hr-lag and $PM_{2.5}$ was strongest at 20-hr lag. Sahlen and others suggested that the risk of STEMI increases within hours of exposure to air pollutants. The study by (Rasche et al., 2018) examined the rapid changes in pollutant (NO_2, NO_x, O_3, PM_{10}) concentrations and the risk of MI. The study was conducted in the city of Jena in Germany from 1st January 2003 to 31st December 2010 with a total of 693 MI cases. A lag time of 0-4days was employed in the generalized linear mixed models (GLMM) to determine the risk of MI and environmental data. Multivariate analysis was also conducted with adjustments made for ambient air temperature, relative humidity, and atmospheric pressure. Rasche and colleagues observed that in the overall population, an increase of NO_x by more than $20\mu g/m^3$ and 8-$20\mu g/m^3$ increases the risk for MI by up to 121% (lag2-3days). In addition, the risk of MI in the overall population was also associated with the rapid changes of ambient NO_2 concentrations whilst the risk of MI increased by 73% with a lag of 1 day. Moreover, similar associations was observed in almost all subgroup analysis. From these findings Rasche and others suggested that the magnitude of increases in nitrogen oxide levels may be an independent risk factor for MI. The study by (Rich et al., 2013) examined whether the fine particles ($PM_{2.5}$) enriched with secondary species (sulphates, nitrates, elemental carbon, organic carbon and ammonium) enhanced the triggering of myocardial infarction (MI) in New Jersey, USA. Rich and colleagues used the time stratified case-crossover approach to make an approximation on the risk of transmural infarction increased tapered element oscillating microbalance (TEOM) levels in the 24 hours before emergency department arrival. Pearson's correlation was employed to determine the relationship between the variables and conditional logistic regression employed to estimate the risk of MI with increased $PM_{2.5}$ concentration with each $10\mu g/m^3$ increase in TEOM $PM_{2.5}$ levels. The authors observed a larger relative odds of MI when $PM_{2.5}$ was laden with species (sulphates, nitrates, and organics) and suggested that air pollution mixtures these days are enhanced by pollutants formed through atmospheric chemistry. (Gardner et al., 2014) investigated whether increased levels of ambient fine particulate pollution ($PM_{2.5}$) in the previous 24 hours was associated with an increased risk in STEMI. The study was conducted in Rochester, New York for the period 1st January 2007 to 31st January 2010. The study population included all STEMI and non-STEMI (NSTEMI) patients who presented to the University of Rochester Medical Center (URMC).Conditional logistic regression was used to examine the association between increased $PM_{2.5}$ concentrations and risk of STEMI or NSTEMI; analysis also accounted for shorter time lags (lag0-11hrs), longer time lags (lag0-95hrs)(lag0-11hrs) and co-morbidities (hypertension, dyslipidemia, diabetes and low-left ventricular ejection fraction). Adjustments was also made for mean temperature and mean relative humidity in the previous 3 hours. Gardener and colleagues observed that each $7.1\mu g/m^3$ increase in $PM_{2.5}$ concentrations was associated with an 18% increase in the risk of STEMI. Hypertensive patients were at a greater risk of STEMI after exposure to fine particulates in the previous hour than those non-hypertensive patients. Gardener and others concluded that pre-existing conditions such as hypertension puts an individual at a greater risk of STEMI when exposed to increased fine particle concentrations. (Argacha et al., 2016) examined the

relationship between exposure to common air pollutants such as PM_{10}, $PM_{2.5}$, O_3 and NO_2 and hospital admissions due to STEMI. Significant comorbidities that may increase the risk of STEMI were also assessed. Conditional logistic regression was employed to analyze the association between air pollution and the risk of STEMI with a 0-day lag time between exposures with the outcome expressed as odds ratios. Risk estimates were calculated for each $10\mu g/m^3$ increase in air pollutants and10^oC increase in ambient temperature. Adjustments were made for ambient temperature and day of the week. Argacha and colleagues observed that the odds for an increase in STEMI was associated with every $10\mu g/m^3$ increase in PM_{10}, $PM_{2.5}$, NO_2 concentrations. The elderly (>75yrs) was more at risk of STEMI. No effect was observed for O_3. These findings prompted the authors to suggest that elderly people were more at risk of STEMI when exposed to PM.(Pope et al., 2015) investigated the effects of increased short-term exposure to $PM_{2.5}$ and acute coronary syndrome (ACS)occurrences including STEMI, NSTEMI and unstable angina. The study was conducted in Utah's Wasatch Front, USA for all patients who received coronary angiography from 10th September 1993 to 15th May 2014. Conditional logistic regression was initially used to determine the relationship between exposure to $PM_{2.5}$ and ACS occurrences with the effect measures expressed as odds ratios per $10\mu g/m^3$ increments of $PM_{2.5}$. Adjustments were made for minimum temperature, dew point temperature and barometric pressure. Pope and colleagues observed that the Odds for ST-segment elevation myocardial infarction increased with each $10\mu g/m^3$ increase in concurrent-day $PM_{2.5}$. From these observations, Pope and other suggests that the triggering of acute coronary events such as ST-segment elevation myocardial infarction may be attributed to increased exposure to fine particulate matter (Liu et al., 2018) examined the effects of short-term exposure to $PM_{2.5}$ on MI hospital admissions. The study was conducted in 26 Chinese cities and STEMI hospital admissions for these cities were obtained from the Hospitalization Summary Reports for the period 1st January 2014 to 31st December 2015. Using the time -stratified case crossover design, the association between pollutant levels and STEMI hospitalizations was analyzed using conditional logistic regression. A1-day lag was used in the analysis to control for the non-linear delayed effects temperature. To evaluate temporal associations, the models were fitted with different lag structures from the current day (lag0) through 5 lag days (lag5). To account for the fact that single-day models may underestimate the association, the associations were estimated with 3-day (lag0-2) and 6-day (lag0-5) moving averages of $PM_{2.5}$ levels. Adjustments were made for other pollutants such as CO, NO_2, SO_2 and public holidays. Liu and colleagues observed that an interquartile range (IQR) increase in $PM_{2.5}$ concentrations ($47.5\mu g/m^3$) at lags 2, 3, 4 and 0-5 days corresponded with 0.6% (95%CI:0.1 -1.1%), 0.8(95%CI:0.3-1.3%), 0.6%(95%CI:0.1-1.1%) and 0.9%(95%CI:0-1.8%) increases in STEMI admissions. Liu and others concluded that 4exposure to short-term increases in $PM_{2.5}$ may increase the risk of STEMI hospitalizations. (Chang et al., 2013) evaluated the short-term effect of exposure to $PM_{2.5}$ and MI (ICD9) hospitalizations. The study was conducted for individuals residing in Taipei city over a 5 year period, from 2006 to 2010. A time-stratified approach was used in this case-crossover study with the stratification of time into separate months to identify referent days falling on the same day of the week within the same month as the index day. A cumulative lag period of up to 2 previous days (i.e. average air pollutant concentrations of the same and previous 2 days). Conditional logistic regression was used to explore the association between the ambient concentrations of $PM_{2.5}$ and the number of MI hospital admissions. Estimates of the associations was expressed as odds ratios and 95% confidence interval with weights equal to the number of hospitalizations on that day. Adjustments was also made for daily average temperature, humidity and barometric pressure as well as for

warm (>23°C) and cool days (<23°). Chang and colleagues observed that for single pollutant models increased MI admissions was significantly associated with an interquartile range increase (IQR) of $PM_{2.5}$ concentrations(17.46 ug/m^3) on both cool (OR=1.05 (95%CI: 1.01-1.09) and warm (OR=1.10(95%CI:1.06-1.15)days. From these findings, the authors suggests that increased levels of $PM_{2.5}$ increases the risk of MI hospitalizations. (Hsieh et al., 2010) examined the relationship between ambient pollutant (SO_2, PM_{10}, NO_2, CO and O_3) levels and MI hospitalizations in Taipei city, Taiwan over an 11 year period from 1996 to 2006. A time-stratified approach was used in this case-crossover study with the stratification of time into separate months to identify referent days falling on the same day of the week within the same month as the index day. A cumulative lag period of up to 2 previous days (i.e. average air pollutant concentrations of the same and previous 2 days). Conditional logistic regression was used to explore the association between the ambient concentrations pollutants and the number of MI hospital admissions. Estimates of the associations was expressed as odds ratios and 95% confidence interval with weights equal to the number of hospitalizations on that day. Adjustments was also made for daily average temperature, humidity and barometric pressure as well as for warm (>23°C) and cool days (<23°). Hsieh and colleagues observed that in single pollutant models, all pollutants except for SO_2 were significantly associated with increased MI admissions on both warm (>23°C) and cold (<23°C) days. In 2 pollutant models concentrations of O_3 and NO_2 in combination with other pollutants were significantly associated with high MI hospital admissions. Hsieh and others suggests that the outcome of this study provides further evidence that increased levels of ambient air pollutants increases the risk of MI hospitalizations. (Cheng, Tsai, & Yang, 2009) evaluated the relationship between concentrations of ambient air pollutants (SO_2, PM_{10}, NO_2, CO, O_3) and MI hospitalizations among individuals who live in Kaosiung over an 11 year period from 1996 to 2006. A time-stratified approach was used in this case-crossover study with the stratification of time into separate months to identify referent days falling on the same day of the week within the same month as the index day. A cumulative lag period of up to 2 previous days (i.e. average air pollutant concentrations of the same and previous 2 days). Conditional logistic regression was used to explore the association between the ambient concentrations of pollutants and the number of MI hospital admissions. Estimates of the associations was expressed as odds ratios and 95% confidence interval with weights equal to the number of hospitalizations on that day. Adjustments was also made for daily average temperature, humidity and barometric pressure as well as for warm (>23°C) and cool days (<23°). Cheng and colleagues observed that in single pollutant models, concentrations of NO_2 (OR=1.19(95%CI: 1.05-1.35), CO (OR=1.18(95%CI: 1.08-1.28) and O_3 (OR=1.18(95%CI: 1.10-1.26) were significantly associated with MI hospital admissions on warm days. On cool days, all pollutants were statistically associated with MI admissions except for O_3. In the two pollutant models, O_3 and CO were significant in combination with each of the other four pollutants on warm days. NO_2 remained statistically significant in all the two pollutant models on cool days. From these findings, Cheng and others suggests that increased concentrations of ambient air pollutants increases the risk of higher frequency of MI hospitalizations. (Bejot et al., 2011) investigated the short-term effects of exposure to ozone on ischemic heart and cerebrovascular disease in Dijon, France from 2001 to 2007. Using a bi-directional case-crossover design analysis daily concentrations of urban O_3 was compared with first-ever, recurrent, fatal and non-fatal ischemic cerebro-vascular events (ICVE) and myocardial infarction. Concentrations of SO_2, NO_2, CO and PM_{10} were used to create bi-pollutant models. Using multivariate logistic modelling, the effects of O_3 exposure were calculated for every 10$\mu g/m^3$ increase in pollutants. Adjustments was also done for all

possible confounders such as weather variables and hypercholesterolemnia. Bejot and colleagues observed that in stratified analysis, O_3 was associated with MI incidence when hypercholesterolemnia was present, OR = 1.111(95%CI: 1.020-1.211) and the strength of the associations increased with increasing number of combined factors. Bejot and others deduced from these findings that even exposure to low concentrations of O_3 can trigger MI especially among individuals with severe vascular risk factors. (Zhang et al., 2016) investigated the relationship between ambient air pollution and emergency department visits due to acute myocardial infarction in Chaoyang District, Beijing, China in 2014. A time-stratified approach was used in this case-crossover study with a lag 0-5days. PM concentrations were compared during the period of patients experiencing AMI (case period) with the times not experiencing AMI (control period). Relative risk of AMI was estimated by comparing PM exposure during case periods and control periods. Adjustments were made for temperature and relative humidity using a natural smooth spline with 3 degrees of freedom in each model. Conditional logistic regression modelling was used to estimate the association between exposure to air pollutants and acute myocardial infarction outcomes. Odds Ratios and 95% confidence intervals scaled to every 10 $\mu g/m^3$ change of particulate matter concentrations was presented as risk of AMI, STEMI and NSTEMI. Single and multiple pollutant models was used to determine the independent/combined effects of air pollutants on AMI outcomes. Zhang and colleagues observed that each 10$\mu g/m^3$ increases in models was used to determine the independent/combined effects of air pollutants on AMI outcomes. Zhang and colleagues observed that each 10$\mu g/m^3$ increases of $PM_{2.5}$ concentration (1-day lagged) was associated with an increased risk of emergency department visits for STEMI, OR=1.05(95%CI: 1.00-1.11). Findings from this study showed a transient effect of short-term exposure to $PM_{2.5}$ on emergency department visits for STEMI and that patients over 65 years of age were most vulnerable. (Hopke et al., 2015) investigated whether fine particulate ($PM_{2.5}$) emissions from different upwind origins were associated with cardiovascular effects among residents in Rochester, New York from 1st January 2007 to 31st December 2010. A time-stratified approach was used in this case-crossover study by contrasting pollutant levels prior to the acute coronary syndrome (case-period) to other periods when the subject did not have an acute coronary syndrome, matched to the case-period by month, weekday, and hour of the day (control periods). To calculate each hourly air mass location for the 24hrs prior to each case or control time period, NOAA hybrid single-particle lagrangian trajectory (HYSPLIT) was employed. HYSPLIT is a system for computing simple air parcel trajectories, dispersion and deposition simulations. Conditional logistic regression was used to examine whether $PM_{2.5}$/STEMI associated was altered by whether the air mass passed through each of the 8 cardinal wind direction (ENE, ESE, SSE, SSE, SSW, WNW, WSW, and NNW) sectors in the previous 24hours. Adjustments were made for mean temperature and mean relative humidity. Hopke and colleagues noted that when air passed through the West-Southwest (WSW) direction anytime in the last 24hours, a statistically significant association between STEMI with each 7.1$\mu g/m^3$ increase in $PM_{2.5}$ concentrations in the previous hour was observed, OR=1.27(95%CI: 1.08-1.22). In addition, the relative odds were largest when the time spent in the WSW was between 8-16hours. From these findings Hopke and others suggest that fine particles from the WSW direction are more potent in triggering STEMIs and that the direction of wind is related to emissions from coal-fired power plants and other industrial sources of the Ohio River valley. (Nuvolone et al., 2011) examined the relationship between air pollutants (PM_{10}, NO_2, CO) and AMI hospitalizations in Tuscany, Italy for the period January 2002 to December 2005. Nuvulone and colleagues used an area-specific case-crossover approach whereby the authors identifies cases and compares each subject's

exposure in a time period just before a case event (the case period) with the subject's exposure at other times (control periods). This study design adjusts for individual characteristics that vary over time such as age, gender, and body mass index (BMI). Conditional logistic regression models were constructed to impute the odds ratios and their 95% confidence intervals. Adjustments were made for variables such as apparent temperature influenza, epidemics and population decreases during vacation periods. To impute immediate and delayed effects different lag patterns were considered using single day lags from lag0 (current day concentration) to lag5 (5 days before the event day). Cumulative lags was also considered by imputing the average between the same day and the previous 5 days (lag0-5) and the average between the previous 3 days and the previous 5 days (lag3-5). Finally, pooled estimates were obtained from the random effects meta-analysis. Nuvolone and others observed that all the pollutants measured were associated with AMI hospitalization with a meta-analytical odds ratio at lag2, 1.013(95%CI: 1.000-1.026) for PM_{10}, 1.022 (95%CI: 1.004-1.041) for NO_2, and 1.007(95%CI: 1.002-1.013). The findings from this study suggests that AMI onset is associated with short-term exposure to air pollutants and that groups like the elderly (>75yrs), females, older patients with hypertension and chronic obstructive pulmonary disease are more vulnerable. The study by (Lin et al., 2013) evaluated the role of gaseous pollutants on AMI hospitalization and mortality in Hong Kong over a 13 year period from 1998 to 2010. Using a time stratified case-crossover design the cases and controls were matched by the day of the week to account for any potential weekly patterns with a 1 day lag. Adjustments was made for temperature, relative humidity and public holidays as confounding factors. Lin and colleagues observed that an in single pollutant models the risk of AMI mortality increased by 4.55% with every interquartile (IQR) increase in NO_2 concentrations. AMI mortality also increased by 2.56% for every interquartile increase in SO_2 concentrations. These findings suggest that in Hong Kong elevated concentrations of SO_2 and NO increases AMI related mortality. Finally the study by (Matsukawa et al., 2014) evaluated the impact of Asian dust (AD) and the incidence of acute myocardial infarction in patients with coronary heart diseases. The study participants were patients admitted into 4 hospitals in the Fukuoka prefecture, Japan from 2003 to 2010. Matsukawa and colleagues used the time-stratified case crossover approach with the case period defined as the day of admission and control periods are the days of the week in the same month of the same year as the case period. Single lag effect was employed (0-5days) and cumulative lag (days 0-1, days 0-5) to account for the persistence of AD, suspended particulate matter (SPM), NO_2 and SO_2 events over a few days. Conditional logistic regressions was employed to examine the association of exposure to AD, SPM, NO_2 and SO_2 the occurrence of AMI. Stratified analysis according to age strata was also performed to examine the effect modification. Matsukawa and colleagues observed significant associations between AD and the incidence of AMI. In addition, significant associations was observed when AD was defined by the SPM (PM<7microns) and the risk of AMI hospitalisation. These findings suggest that exposure to AD prior to the onset of symptoms is associated with the incidence of AMI

4.8 Key features and conclusions of the studies included in the meta-analysis

Table 8: Key features of each study

Author	Location	Description of study and main Conclusions
Cheng, 2009	Kaohsiung, Taiwan	Evaluated the relationship between concentrations of ambient air pollutants (SO_2, PM_{10}, NO_2, CO, O_3) and MI hospitalizations among individuals who live in Kaosiung over an 11 year period from 1996 to 2006.Findings from this suggests that increased concentrations of ambient air pollutants increases the risk of higher frequency of MI hospitalizations
Hsieh, 2010	Taipei, Taiwan	Examined the relationship between ambient pollutant (SO_2, PM_{10}, NO_2, CO and O_3) levels and MI hospitalizations in Taipei city, Taiwan from 1996 to 2006. Findings from this study suggests that the outcome of this study provides further evidence that increased levels of ambient air pollutants increases the risk of MI hospitalizations
Nuvulone, 2011	Tuscany, Italy	Examined the relationship between air pollutants (PM_{10}, NO_2, CO) and AMI hospitalizations in Tuscany, Italy for the period January 2002 to December 2005.Findings from this study suggests that AMI onset is associated with short-term exposure to air pollutants and that groups like the elderly (>75yrs), females, older patients with hypertension and chronic obstructive pulmonary disease are more vulnerable
Wang, 2016	Pudomng district, Shanghai, China	Assessed the variations in acute myocardial infarction (AMI) hospital admissions relative to air pollutant levels between 1st November 2013 and 27 April 2014 in Shanghai, China. Findings from this study suggests that an increased risk in AMI is associated with short-term exposure to moderate – serious pollutant levels
Weichenthal, 2017	British Columbia, Canada	Examined if hospital admissions for myocardial infarction (MI) was associated with short-term changes in outdoor $PM_{2.5}$. The study was conducted in 3 regions in British Columbia from 2008 to 2015. Findings from this study suggests that a short-term change in outdoor $PM_{2.5}$ levels increases the risk of MI
Hopke, 2015	Rochester, New York, USA	Investigated whether fine particulate ($PM_{2.5}$) emissions from different upwind origins were associated with cardiovascular effects among residents in Rochester, New York from 1st January 2007 to 31st December 2010. Findings from this study suggests that fine particles from the WSW direction are more potent in triggering STEMIs and that the direction of wind is related to emissions from coal-fired power plants and other industrial sources of the Ohio River valley
Chang, 2013	Taipei, Taiwan	Evaluated the short-term effect of exposure to $PM_{2.5}$ and MI hospitalizations in Taipei city from 2006 to 2010. Findings from this study suggests that increased levels of $PM_{2.5}$ increases the risk of MI hospitalizations
Evans, 2016	Monroe County New York,USA	Examined the onset of STEMI after exposure to wood smoke and traffic pollution in Monroe County, New York from 1st January 2007 to 30 September 2012.Finidngs from this study suggests the increased levels of $PM_{2.5}$ increases the odds of STEMI
Pope, 2015	Utah's Wasatch Front, USA	Investigated the effects of increased short-term exposure to $PM_{2.5}$ and acute coronary syndrome (ACS) occurrences including STEMI, NSTEMI and unstable angina. Findings from this study suggests that the triggering of acute coronary events such as ST-segment elevation myocardial infarction may be attributed to increased exposure to fine particulate matter
Argacha, 2016	Belgium	Examined the relationship between exposure to common air pollutants such as PM_{10}, $PM_{2.5}$, O_3 and NO_2 and hospital admissions due to STEMI. Findings from this study suggests that elderly people were more at risk of STEMI when exposed to PM

Zhang, 2016	Chaoyang, Beijing China	investigated the relationship between ambient air pollution and emergency department visits due to acute myocardial infarction in Chaoyang District, Beijing, China in 2014.Findings from this study showed a transient effect of short-term exposure to $PM_{2.5}$ on emergency department visits for STEMI and that patients over 65 years of age were most vulnerable
Rasche,2018	Jena, Germany	Examined the rapid changes in pollutant (NO2, NOx, O3, PM10) concentrations and the risk of MI in the city of Jena in Germany from 1st January 2003 to 31st December 2010. Finings from this study suggests that the magnitude of increases in nitrogen oxide levels may be an independent risk factor for MI
Sahlen, 2018	Stockholm, Sweden	examined the association of short-term risk of STEMI after exposure to of air pollutants. Findings from this study suggests that the risk of STEMI increases within hours of exposure to air pollutants.
Bejot, 2010	Dijon, France	Investigated the short-term effects of exposure to ozone on ischemic heart and cerebrovascular disease in Dijon, France from 2001 to 2007.Findings from this study suggests even exposure to low concentrations of O_3 can trigger MI especially among individuals with severe vascular risk factors
Rich, 2013	New Jersey, USA	Examined whether the fine particles (PM2.5) enriched with secondary species (sulphates, nitrates, elemental carbon, organic carbon and ammonium) enhanced the triggering of myocardial infarction (MI) in New Jersey, USA. Findings from thi study suggests that air pollution mixtures these days are enhanced by pollutants formed through atmospheric chemistry
Berglind,2010	Sweden	Explore the influence of short-term exposure to air pollution immediately preceeding myocardial infarction in Stockholm from 1993 to 1994.Findings from this study suggests that there is no risk of MI onset at moderately elevated air pollution levels
Gardner, 2014	Rochester, New York, USA	Investigated whether increased levels of ambient fine particulate pollution ($PM_{2.5}$) in the previous 24 hours was associated with an increased risk in STEMI. Findings from this study suggests that pre-existing conditions such as hypertension puts an individual at a greater risk of STEMI when exposed to increased fine particle concentrations
Chiu, 2017	Taipei, Taiwan	investigated the short-term effects of ambient O3 on the daily hospitalizations of MI in Taipei, Taiwan from 2006 to 2010. Findings from this study suggests that short-term exposure to O_3 concentrations can increase the MI hospital admissions on both warm and cool days
Matsukawa, 2014	Fukuoka, Japan	Evaluated the impact of Asian dust (AD) and the incidence of acute myocardial infarction in patients with coronary heart diseases. The study participants were patients admitted into 4 hospitals in the Fukuoka prefecture, Japan from 2003 to 2010.Findings from this study suggests that exposure to AD prior to the onset of symptoms is associated with the incidence of AMI
Lin, 2012	Hong Kong	Evaluated the role of gaseous pollutants on AMI hospitalization and mortality in Hong Kong over a 13 year period from 1998 to 2010.Findings from this study suggest that in Hong Kong elevated concentrations of SO2 and NO increases AMI related mortality

4.9 Characteristics of studies included in the meta-analysis

Table 9: Characteristics of included studies

Author	Location	Exposure	Outcome	Study design	Lag	OR	Lower_CI	Upper_CI	N	Quality Score
Cheng, 2009	Kaohsiung, Taiwan	SO_2, NO_2, PM_{10}, CO, O_3	AMI Hospitalization	Case-crossover	2 days	1.41	1.27	1.57	9349	3
Hsieh, 2010	Taipei, Taiwan	SO_2, NO_2, PM_{10}, CO, O_3	AMI Hospitalization	Case-crossover	0	1.1	1.05	1.15	23420	3
Nuvulone, 2011	Tuscany, Italy	CO,NO_2, PM_{10}	AMI Hospitalization	Case-crossover	0, 1,2,3,4,5	1.013	1	1.026	11450	3
Wang, 2016	Pudomng district, Shanghai, China	$PM_{2.5}$, PM_{10},SO_2,NO_2, CO	AMI Hospitalization	Case-crossover	0	1.16	1.03	1.29	972	3
Weichenthal, 2017	British Columbia, Canada	$PM_{2.5}$	AMI Hospitalization	Case-crossover	3 day mean					
Hopke, 2015	Rochester, New York, USA	$PM_{2.5}$	STEMI hospitalization	Case-crossover	3-24hrs	0.99	0.8	1.22	338	3
Chang, 2013	Taipei, Taiwan	$PM_{2.5}$,PM_{10},CO,NO_2,SO_2,O_3	AMI Hospitalization	Case-crossover	0	1.04	1	1.08	14353	3
Evans, 2016	Monroe County New York,USA	Wood smoke (Delta C), traffic pollution(BC),$PM_{2.5}$, NO_2, SO_2, O_3,CO	STEMI hospitalisations	Case-crossover	1 hr	1.17	0.99	1.39	362	3
Pope, 2015	Utah's Wasatch Front, USA	PM_{10}, $PM_{2.5}$	AMI Hospitalization	Case-crossover	1 day	1.02	0.97	1.08	1274	3
Argacha, 2016	Belgium	PM_{10},$PM_{2.5}$,O_3,NO_2	STEMI hospitalisations	Case-crossover	0	1.026	1.005	1.048	11428	2

Zhang, 2016	Chaoyang, Beijing China	$PM_{2.5}$, PM_{10},NO_2, SO_2,O_3, CO	STEMI	Case-crossover	1 day	1.05	1	1.11	2749	3
Rasche,2018	Jena, Germany	NO_2, PM_{10}, O_3	AMI Hospitalization	Case-crossover	1-24hrs	2.21	1.19	4.08	693	3
Sahlen, 2018	Stockholm, Sweden	NO_2,SO_2,O_3,PM_{10}, $PM_{2.5}$	AMI Hospitalization	Case-crossover	0	1.026	1.001	1.054	14601	2
Bejot, 2010	Dijon, France	O_3	AMI Hospitalization	Case-crossover	3 day	1.111	1.02	1.211	913	2
Rich, 2013	New Jersey, USA	$PM_{2.5}$	AMI Hospitalization	Case-crossover	0	1.13	1	1.27	1562	3
Gardner, 2014	Rochester, New York, USA	$PM_{2.5}$	AMI Hospitalization	Case-crossover	0 days	1.17	0.98	1.4	338	3
Chiu, 2017	Taipei, Taiwan	O_3	AMI Hospitalization	Case-crossover	0 days	1.07	1.02	1.12	14353	3
Matsukawa, 2014	Fukuoka, Japan	Asian dust, suspended particle matter, NO2,SO2	AMI hospital admissions	Case-crossover	4 days	1.43	1.03	1.98	3068	3
Lin, 2012	Hong Kong	PM10,NO2,SO2,O3,CO	AMI Hospitalization		1 day	1.0455	1.017	1.0748	24,432	2
Berglind,2010	Sweden	PM10	AMI Hospitalization	Case-crossover	0 day	0.989	0.836	1.166	660	3

4.10 Forest Plots

4.10.1 Overall analysis

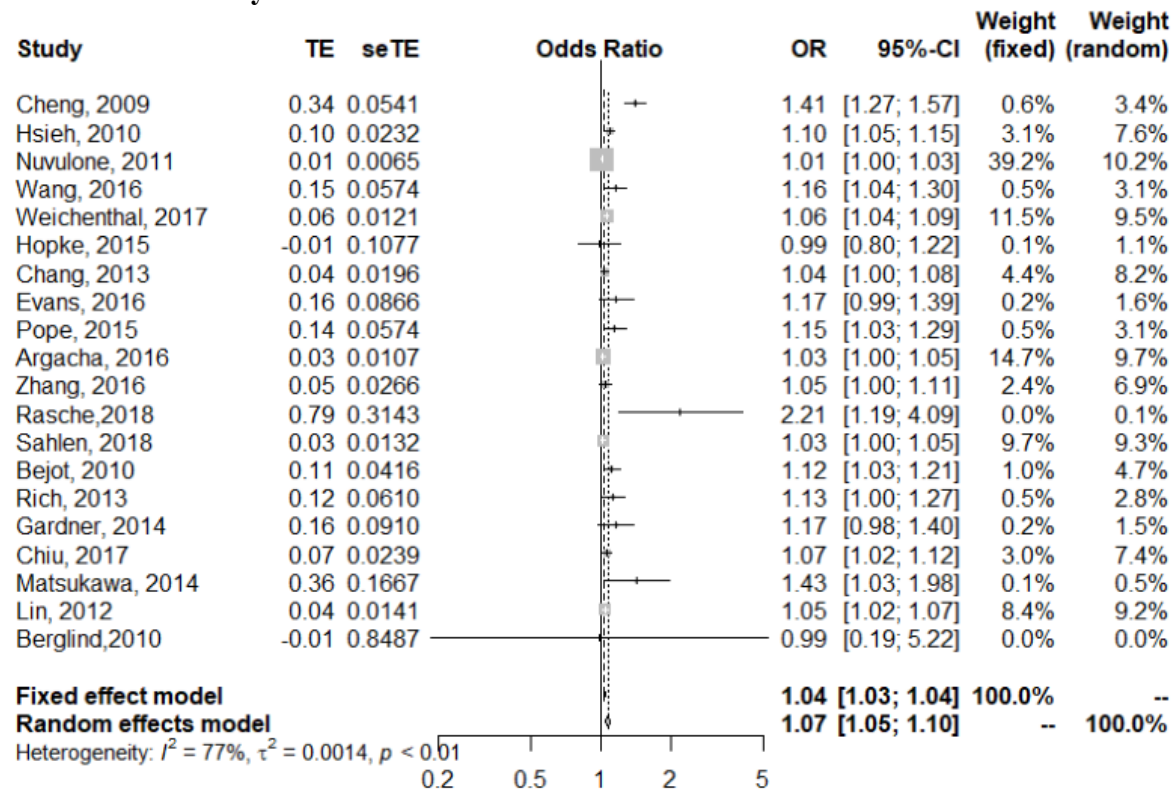

Figure 31: Forest plot of pooled estimates of the overall effects of exposure to air pollutanst and risk of AMI hospitalizations

Figure 30 illustrates the forest plot showing the summary estimates of 20 included studies on the association between air pollutants and AMI hospitalization. Each study is represented by a horizontal line in the plot (from left to right). The effect estimate of each study in this meta-analysis was presented as Odds Ratio (e.g. the study by Cheng, 2009 yielded a weighted average, OR=1.41 (95%CI: 1.27-1.57). In the graph the grey box represents the weight (odds) of the study i.e. the bigger the box the bigger the weight and the smaller the box the smaller the weight. Each horizontal line emanating out of the box represents the 95% Confidence Interval of each study. The diamond at the bottom of the graph indicates the weighted average of all studies and the tips of the diamond represent the 95% confidence interval

Evaluating the statistical heterogeneity of the studies using a variation of chi-squared test or Q Statistic test yielded the result, 84.26 (degrees of freedom 19; p<0.001). To quantify this heterogeneity the I^2 test was 77% indicating a high degree of heterogeneity. Since the studies are heterogeneous they form part of a large "universe of studies and the results are part of a randomly distributed effect measure. The random effects model pooled estimated suggested a significant positive association between exposure to common air pollutants and risk of AMI hospitalization, (OR = 1.0741 (95%CI: 1.0490 – 1.0998). In addition, the fixed effect model pooled estimate also suggested a significant positive association between exposure to common air pollutants and risk of AMI hospitalization, (OR = 1.363 (95%CI: 1.0280 – 1.0447).

4.10.2 Subgroup analysis

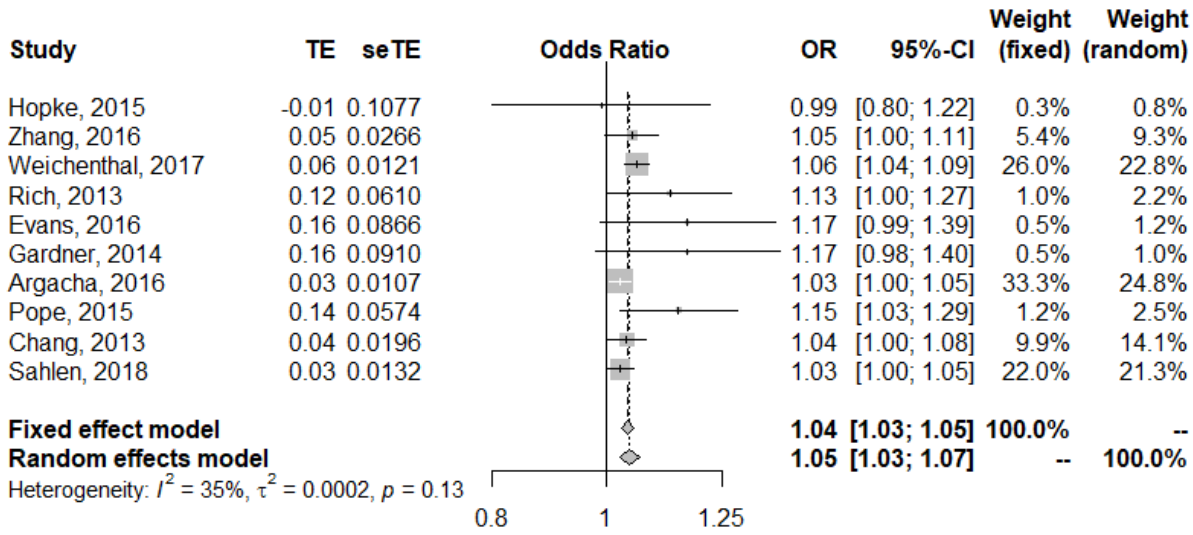

Figure 32: Forest plot of the pooled estimates for the subgroup analysis on the risk of AMI hospitalization associated due to each 5-10ug/m^3 increments in $PM_{2.5}$ concentrations

Figure 31 illustrates the forest plot showing the summary estimates of 8 studies in estimating the risk of AMI hospitalization associated with at least each 5-10µg/m^3 increases in $PM_{2.5.}$ Each study is represented by a horizontal line in the plot (from left to right). The effect estimate of each study in this meta-analysis was presented as Odds Ratio (e.g. the study by Hopke, 2015 yielded a weighted average, OR=0.99 (95%CI: 0.80-1.22). In the graph the grey box corresponds to the sample size of the study i.e. the bigger the box the bigger the sample size and the smaller the box the smaller the sample size. Each horizontal line emanating out of the box represents the 95% Confidence Interval of each study. The 2 diamonds at the bottom of the graph corresponds to the fixed effects and random effects estimates and the tips of the diamond represents the 95% confidence interval.

Evaluating the statistical heterogeneity of the studies using a variation of chi-squared test or Q Statistic test yielded the result, 13.91 (degrees of freedom 9; p = 0.1256). To quantify the heterogeneity, the I^2 test was 35.3% indicating a low degree of heterogeneity. Since the studies are show a low degree of heterogeneity the random effects model pooled estimates suggested a significant positive association between exposure to common air pollutants and risk of AMI hospitalization, (OR = 1.0458 (95%CI: 1.0267 – 1.0654); p<0.001. In addition, the random effects model pooled estimate also suggested a significant positive association between exposure to common air pollutants and risk of AMI hospitalization, (OR = 1.07 (95%CI: 1.02 – 1.12).

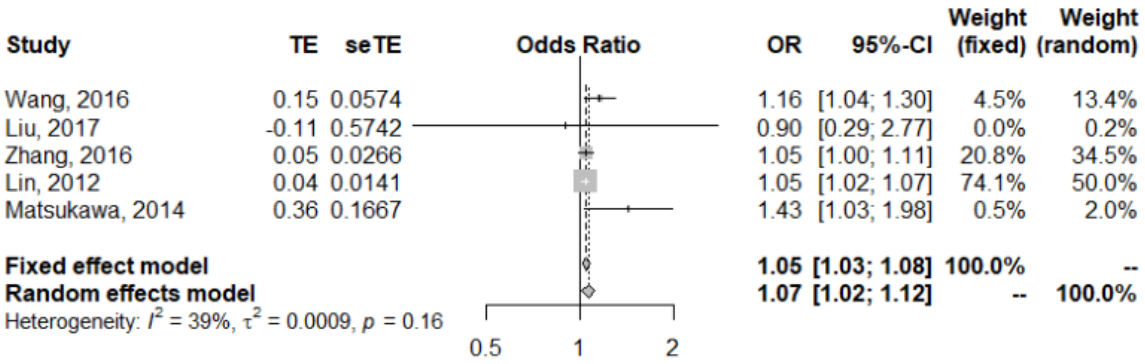

Figure 33: Forest plot of the pooled estimates in the subgroup analysis on the effects of exposure to air pollutants and risk of AMI hospitalizations in Western Pacific Region countries

Figure 32 illustrates the forest plot showing the summary estimates of 5 studies in WHO Western Pacific regional countries on the association between air pollutants and AMI hospitalization. Each study is represented by a horizontal line in the plot (from left to right). The effect estimate of each study in this meta-analysis was presented as Odds Ratio (e.g. the study by Wang, 2016 yielded a meta-analytic result, OR=1.16 (95%CI: 1.04-1.30). In the graph the grey box corresponds to the sample size of the study i.e. the bigger the box the bigger the sample size and the smaller the box the smaller the sample size. Each horizontal line emanating out of the box represents the 95% Confidence Interval of each study. The 2 diamonds at the bottom of the graph corresponds to the fixed effects and random effects estimates and the tips of the diamond represents the 95% confidence interval.

Evaluating the statistical heterogeneity of the studies using a variation of chi-squared test or Q Statistic test yielded the result, 6.55 (degrees of freedom 4; p<0.1615). To quantify the heterogeneity, the I^2 test was 39% indicating a moderate degree of heterogeneity. Since the studies show a moderate heterogeneity the fixed effects model pooled estimates suggested a significant positive association between exposure to common air pollutants and risk of AMI hospitalization, (OR = 1.05 (95%CI: 1.03 – 1.08); p<0.0001. In addition, the random effects model pooled estimate also suggested a significant positive association between exposure to common air pollutants and risk of AMI hospitalization, (OR = 1.07 (95%CI: 1.02 – 1.12).

4.11 Funnel plots

4.11.1 Overall analyses

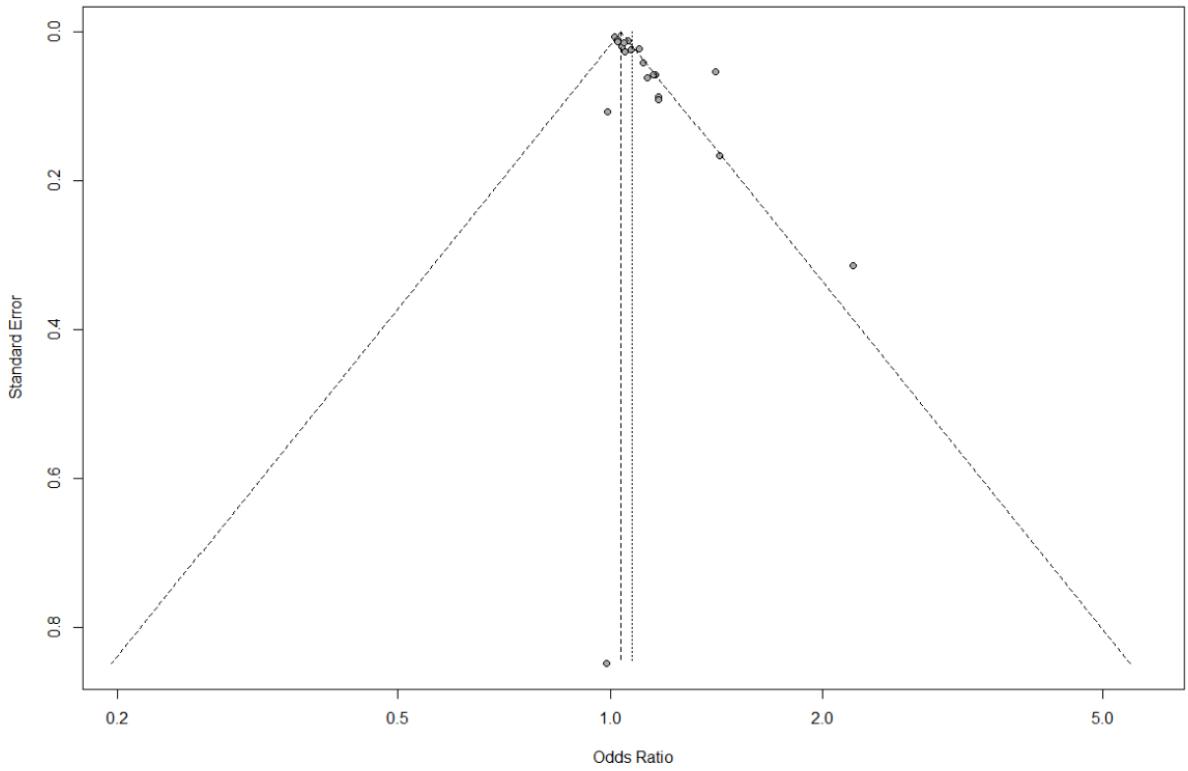

Figure 34: Funnel plot on the overall analysis of the association between exposure to common air pollutants and risk of AMI hospitalization

Figure 35 illustrates the funnel plot to detect publication bias for the studies included in the meta-analysis. This is a plot where the Y-axis consists of the inverse variance and the X-axis consists of the effect estimate and the points represents the studies themselves. The ends of the funnel represent the 95% confidence intervals with the left indicating the lower confidence interval and the right end indicating the upper confidence interval. The vertical line represents the summary effect of the meta-analysis. In this way, studies that have wide variance (and therefore low powered and small sample sizes) should be present in the lower part of the graph and studies that have large sample size and therefore low variance will be present in the upper part of the graph and the distribution of the studies should follow the shape of a funnel. If all the quadrants of the funnel are equally represented by studies, then it would be presumed that a wide range of studies have been captured that have both large and small sample sizes, as well as studies that have reported negative results or studies that have reported positive results or studies that have failed to refute the null hypothesis.

However, in Figure 35 most of the studies are huddled on the top, it signifies that most of the studies have large sample size or with low variance and studies that are equivocal and studies that have negative results have been missed. This signifies that all studies have not been captured for a meta-analysis and this thesis is biased towards studies that have only reported estimates on the basis of large sample sizes.

4.11.2 Subgroup analyses for short-term exposure to 5-10µg/m^3 increments of $PM_{2.5}$

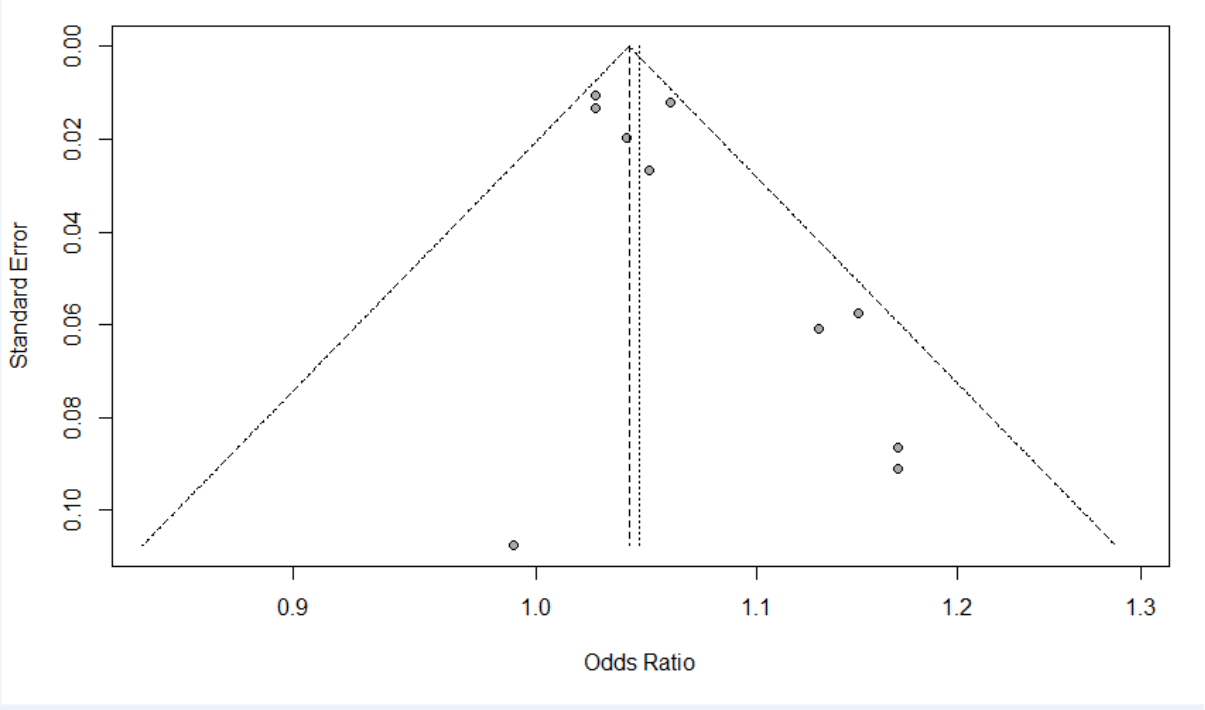

Figure 35: a funnel plot on the subgroup analysis on the risk of AMI hospitalization associated with at least each 5-10µg/m^3 increments in PM2.5 concentrations

Illustrates the funnel plot to detect publication bias for the studies included in the meta-analysis. Begg's funnel plot as illustrated above indicated evidence suggestive of publication bias. Most of the studies are huddled to the top as well as to the right of the funnel which suggests that the meta-analysis relied mostly on studies that had large standards errors and seem to report larger effect sizes on average. In addition, the grapg suggests that studies with small or opposite effects seem to be missing from the meta-analysis

4.12 Publication Bias

The studies selected for this meta-analysis was dependent on the nature and direction of their results. Hence, studies with more significant results were perceived as important. In addition, the Begg's plot shown in the overall pooled estimates as well as in the subgroup analyses suggest that studies that were smaller in size or had equivocal results were missed. Failure to include all relevant studies in this meta-analysis is not solely attributed to publication bias as other sources of bias exist and are described collectively as reporting bias. Publication of studies with equivocal results is at times influenced by the study size, funding source or research group (Sedgwick, 2013).

4.13 Predicted risk of AMI hospitalization in Lautoka city based on daily air quality assessment

The pooled estimates in both the overall and subgroup analysis show significant association between exposure to criteria pollutants and the risk of AMI hospitalization. Based on the pooled estimates from both the overall and subgroup meta-analysis and the air quality data (refer to Tables 5 & 6), it is estimated that if the concentrations of particulate matter (PM_{10} and $PM_{2.5}$) persist, there will be an increase in hospital admissions due to acute myocardial infarction. In addition, in a week, high admission rates due to heart disease should be expected on Thursday and Fridays after exposure to both high PM_{10} levels and even low $PM_{2.5}$ concentrations. Additionally, on a day with poor air quality levels, it is estimated that high rate of heart disease related emergency hospital admissions may occur after exposure to high pollutant levels during peak traffic hours.

CHAPTER 5

DISCUSSIONS

5.1 Summary

This chapter highlights the output obtained from the methodologies used. The mean concentrations of the pollutant PM10 during this study was observed to be significant. The mean concentrations of PM10 in the duration of the study and the imputed daily mean concentrations both exceeded the WHO recommended guideline value. Even though the mean concentrations of PM2.5 were relatively low. Its presence even in low concentrations as observed in this study is still of great health concern, in particular on the objectives of this study. Concentrations of other pollutants were insignificant but their presence is worth noting and is deliberated further in the next chapter. The concentrations of both PM10 and PM2.5 during the hours of a day suggests that contributions from vehicles is quite significant. The systematic review and meta-analysis provided some evidence in both the overall and subgroup analyses of the relationship between short-term exposure to air pollutants and risk of AMI hospitalizations. There was evidence of high heterogeneity and low heterogeneity in both the overall and subgroup analyses respectively. Publication bias was present in both overall and subgroup analyses and some factors that may contribute to this biases is discussed in the next chapter.

5.2 Introduction

Despite the growing epidemiological research and evidence on the health impact of air pollution for the past two decades, there has been very little research undertaken in Fiji and surprisingly, none has been done in Lautoka. This thesis will be the first study to examine the ambient air quality in the Lautoka Central Business District and its potential health impacts, specifically, risk of hospitalizations due acute myocardial infarction. To achieve this, the principle objectives that were developed are:

1. Determining the ambient air quality in the Lautoka CBD and its potential health implications
2. Conducting a meta-analysis on the association between ambient air quality and risk of hospitalization due to myocardial infarction
3. On the basis of the meta-analysis and air quality data, I will develop predictive models on the risk of hospitalization due to acute myocardial infarction for Lautoka.

Significant findings from the results are discussed in relation to the three principle objectives and in the context of international research and literature. Also, extrapolation on the implications of the findings on the risk of AMI hospitalization in Lautoka are also discussed in this chapter. Limitations of this research and future directions are also elaborated herein.

5.3 Ambient air quality in the Lautoka CBD

5.3.1 Mean concentrations of Particulate Matter (PM_{10} and $PM_{2.5}$)

This study showed that particulate matter (PM) levels, in particular $PM_{10,}$ was quite significant in the ambient air quality data gathered from the Lautoka Central Business District. The recommended 24hr mean concentrations for particulate matter with an aerodynamic diameter < 10microns (PM_{10}) is 50μg/m^3 (WHO, 2006). The daily mean PM_{10} concentrations recorded for the Lautoka CBD was 64.15μg/m^3 which was in excess of 14.15μg/m^3 when compared with the WHO recommended guideline (50 μg/m$^{3)}$. Although these PM_{10} concentrations were high, other tropical cities like Kaohsiung in Taiwan have observed higher concentrations as observed in the study by (Cheng et al., 2009). Despite this high concentrations, the WHO's Global Health Observatory (GHO) data reported that for urban areas mean concentrations of PM_{10} are expected to be within the range of less than 10μg/m^3 to over 200μg/m^3. In addition, in a typical day (hours of the day) the analyzed data observed an increase in PM_{10} concentrations in the morning commencing after 5 am and was at its peak between 6am to 8am and in the afternoon PM_{10} concentrations started to increase exponentially after 3pm and peaked at 8pm before decreasing again(ref to Fig. 1). The daily mean $PM_{2.5}$ concentrations observed for Lautoka CBD during this study was 9.34μg/m^3 and was well below the recommended WHO-24hr mean air quality guideline of 25 μg/m^3. In comparison, the WHO's Global Health Observatory (WHO, 2018) data reported that the urban mean concentrations of fine particulate matter ($PM_{2.5}$) in Fiji is 10.5μg/m^3 and the expected range of $PM_{2.5}$ concentrations in urban areas is between less than 10μg/m^3 to over 100μg/m^3. In a typical day (24 hours), it was observed that an increase in $PM_{2.5}$ concentrations occurred after 5am and was at its peak at 8am. Another increase in $PM_{2.5}$ concentrations occurred in the afternoon starting at 4 pm and was at its peak at 8m before decreasing (refer to Fig 2). The two periods (morning and afternoon) whereby high concentrations of particulate matter (PM_{10} and $PM_{2.5}$) occurred are when the volume of vehicles (including buses and trucks) are at its highest in the city and this is a norm for most urban centers in Fiji. In these two periods, buses, trucks, other public transport and private vehicles converge into the city to pick and drop workers and students which could result in high particulate emission from these vehicles. Pearson's correlation also indicated a strong (positive) linear relationship between the two pollutants suggesting the existence of multiple sources apart from vehicle emissions. In terms of the pollutant sources and volume emitted, the Department of Environment in Fiji in 2013 had generally agreed that emissions from vehicles were the major source of air pollution in Fiji.

Fine particles, namely $PM_{2.5}$ are commonly associated with emissions from combustion or secondarily formed aerosol particles, as opposed to coarse particles (< 10microns) PM_{10} which is commonly associated with wind-blown dust, crushing and grinding actions or resuspension by vehicle movement(WHO, 2009). Findings from Studies by (Kleeman et al., 2000; Bagley, 1996) concurred with this statement and their studies found that motor vehicles are among the major contributors of particulate matter emissions in the urban atmosphere. In comparison, major cities in the neighbouring countries of Australia and New Zealand have maintained low levels of PM_{10} and $PM_{2.5}$ (Barnett et al., 2005). This is despite the fact that these cities have larger populations, high number of vehicles and a thriving agricultural and industrial economy. Vehicle emissions along roads in urban areas are released in close proximity to human receptors, giving reduce opportunity for the atmosphere to dilute the emissions. Furthermore, concentrations of pollutants are significantly enhanced by the fact that many roads in cities have buildings alongside. As a result, these building reduces the effect of wind speed at the source of emissions by as much as an order of magnitude relative to that on an open road (Colvile, Hutchinson, & Warren, 2002). This observations by Colvile and others on road traffic

and effects of urban air quality on human health is consistent with the layout of the Lautoka CBD and presumably the exposure methods of the public to air contaminants would be quite similar.

The decrease in particulate pollution in between the peak periods for the Lautoka city can be attributed to the low volume of traffic movement. This is also the time when most of the workers and students are at their workplaces or in schools respectively. Despite this low volume of traffic PM_{10} concentrations were still high ($30\mu g/m^3$ - $70\mu g/m^3$) late into the night and in the early hours of the morning. This could be attributed to other potential particulate sources of notably from power generation, industrial combustion, windblown pollens, and agricultural activities and from open burning of wastes (Brook et al., 2004). Suffice to note that the duration of this study coincided with the sugar cane crushing season in Lautoka. In the crushing season, operation of the mill is on a 24 hour basis unless there is a mechanical breakdown. Apart from the emissions from the operation of the sugar mill, the burning of sugar cane farms to alleviate access for harvesting and to reduce volume of waste material remains an issue of concern in Fiji. In a study by (Mnatzaganian, Pellegrin, Miyamura, Valencia, & Pang, 2015) on the association between sugar cane burning and acute respiratory illnesses, the authors found a significant association between the incidence of respiratory distress and the burning of sugar cane farms. From their findings, Mnatzaganian and others concluded that the more sugar cane farms that are burnt the higher the incidence of respiratory distress are likely to occur.

The health implications of particulate air pollution has been consistently shown in epidemiological studies. Not only does these studies show an association between particulate air pollution and the exacerbation of illnesses in people with respiratory disease but also increases in the number of deaths from cardiovascular and respiratory disease among the elderly (Seaton et al., 1995). This was observed in the study by (Brunekreef & Holgate, 2002) where high ambient particulate (PM_{10} and $PM_{2.5}$) pollution was associated with mortality and hospital admissions due to respiratory and cardiovascular diseases in both short-term and long-term studies. Epidemiological studies from around the world have consistently demonstrated that both short-term and long-term exposures to particulate matter are associated with a host of cardiovascular diseases, including myocardial infarction, heart failure, arrhythmias, strokes and increased cardiovascular mortality. Significantly, evidence from cellular/ toxicological experiments, animal and human exposures and human panel studies have indicated several mechanisms by which particle exposure may trigger acute events as well as expedite the chronic development of cardiovascular diseases (Brook, 2008).

5.3.2 Mean concentrations of Ozone

The mean concentrations for ozone during the study period was very minimal as shown in Table 4 and are well below the WHO guideline values (WHO, 2006). Even though the concentrations of O_3 as negligible, the observations made on the mean concentrations within the hours of a day was characteristic of its nature. Tropospheric ozone or ground-level ozone (O_3) is a secondary pollutant which is created by chemical reactions between oxides of nitrogen and volatile organic compounds (VOC) in the presence of sunlight. It reaches unhealthy levels on hot sunny days in urban environments (USEPA, n.d.). In addition, it is usually at the highest concentration in the afternoon or early evening (NSW_Health, 2017). This phenomenon was observed in this study when ozone concentrations were at its peak in the midday between 11am and 12pm (refer to Figure 5). In the afternoon, ozone concentrations increased again between 4pm and 6pm. Temperature levels were also high during the ozone peak periods suggesting a relationship between the two variables. Moreover, Pearson's correlation validated the existence of a linear (positive) relationship between the two variables, $r = 0.53$ and suggests that

meteorological parameters like temperature can influence the concentrations of ozone. It can also be deduced that the ambient air in the Lautoka CBD contains oxides of nitrogen and volatile organic compounds concentrations contributing to the increases of tropospheric ozone. Apart from causing respiratory diseases, recent evidence have linked O3 with cardiovascular diseases as well. For example, the study by (Bejot et al., 2011) investigated the effects of short-term exposure to O_3 and its impact on ischemic heart and cerebrovascular disease. This case crossover study compared daily levels of urban O_3 pollution and the incidence of first-ever, recurrent, fatal and non-fatal ischemic cerebro-vascular events (ICVE) and myocardial infarction. The authors of this study observed that apart from ICVE, there was a marginally significant association between low levels of O_3 and myocardial infarction with 1-day lag, OR=1.147(95%CI: 0.999-1.318). In addition, when adjusting for confounders like hypercholesterolemia an association between myocardial infarction and O_3 was observed, OR=1.111(95%CI: 1.020-1.211). From these findings, the authors suggested that recurrent Myocardial Infarction could be triggered by short-term exposure to even low O_3 concentrations, specifically for individuals with severe vascular risk factors. Other studies such as that by (Cheng, Tsai, & Yang, 2009) where they investigated the association between air pollutant levels and increased hospital admissions for myocardial infarction in a tropical city (Kaohsiung) in Taiwan yielded similar findings. Cheng and colleagues observed that on warm days (>25oC) concentrations of O_3 were positively associated with increased daily hospitalizations due to myocardial infarction. The authors of this study concluded that increased concentrations of pollutants such as O_3 increases the risk of higher frequency of hospital admissions due to myocardial infarction. The findings from these studies suggest that even in low concentrations and on warm days, O_3 has the potential to cause adverse effects on the heart leading to increased hospitalizations

5.3.3 Mean concentrations of Carbon Monoxide

The average concentrations for the air pollutant carbon monoxide in this study was similar to zone. It is safe to presume that the mean CO concentrations in the Lautoka CBD was very minimal (with the maximum recorded daily concentration of 0.63ppb) when comparing it with the USEPA standards (USEPA, 2018). The primary sources of carbon monoxide in the ambient air around urban areas are commonly from cars, trucks and other vehicles or machinery that burn fossil fuels. Carbon monoxide binds with haemoglobin with an affinity more than oxygen to form carboxyhemoglobin in the red blood cells and interferes with the release of oxygen at tissue level (Brook et al., 2004). Even though the carbon monoxide concentrations observed during this study was very low, it is imperative to understand the impact it may have on the cardiovascular health of the Lautoka populace. Hence, epidemiological findings in the associations between NO_2 and acute myocardial infarction is thoroughly discussed hereafter For instance, in a study by (Allred et al., 1989) where they investigated the short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease, they observed a decrease of 5.1%(90CI:1.5 to 8.7%; p =0.02) and a decrease of 12.1%(90%CI:9.0-15.3%; p<0.000-1) in the length time to a threshold ischemic ST-segment change (ST end point) after carbon monoxide exposures that produced carboxyhemoglobin levels of 2% and 3.9% respectively. In the addition, the time for the onset of angina decreased by 4.2% at the 2% carboxyhemoglobin level and by 7.1% at the 3% carboxyhemoglobin level. Moreover, Allred and others concluded that low concentrations of carboxyhemoglobin aggravates myocardial ischemia during exercise in subjects with coronary heart disease. A similar study was conducted by (ANDERSON, 1973) whereby the author investigated the cardiovascular effects of exposure to low concentrations of CO on 10 men with stable angina pectoris. In this study the participants initially breathed air, then breathed air with 50ppm CO or 100 ppm CO for 4hours on 5 consecutive days. Once they have been exposed to breathing CO a standard treadmill exercise electrocardiogram (ECG) was recorded and the time of onset

and duration of pain was recorded. The results of this study showed that the average carboxyhemoglobin (COHb) increased after breathing air with 50ppm CO (1.3% rise) and a 4.5% rise after 100ppm CO. In addition, there was no prolonged pain after breathing air with 50ppm CO but a duration of pain was significantly continuous after breathing air with 100ppm carbon monoxide. Findings from this study suggested that low levels of carbon monoxide can cause decrease exercise tolerance and worsening of myocardial ischemia in patients with angina pectoris. Other studies such as that by (Cheng, Tsai, & Yang, 2009) where they investigated the association between air pollutant levels and increased hospital admissions for myocardial infarction in a tropical city (Kaohsiung) in Taiwan yielded similar findings. Cheng and colleagues observed that on warm days (>25ºC) concentrations of CO were positively associated with increased daily hospitalizations due to myocardial infarction. The authors of this study concluded that increased concentrations of pollutants such as CO increases the risk of higher frequency of hospital admissions due to myocardial infarction. In another study by (Stieb et al., 2009), the authors examined the associations between common air pollutants and respiratory or cardiac conditions for cities in Canada. Stieb and colleagues observed that an increase in 0.7ppm CO was associated with a 2.1% increase in hospital visits due to myocardial infarction/angina. From these findings, the authors concluded that daily mean levels of CO was most reliable for emergency department visits due to cardiac conditions. Similar findings were observed for the pollutant NO_2 in this study by Stieb and others.

5.3.4 Mean concentrations of Nitrogen dioxide

Nitrogen dioxide concentrations in the Lautoka CBD was similar to that observed for carbon monoxide and ground level-ozone in that it was very low. Despite the low levels of NO_2, its occurrence indicates emissions from motor vehicles and nearby industrial processes around Lautoka City. As a developing nation, understanding these emissions sources and how it may impact the cardiovascular health of its populace is warranted, hence, epidemiological findings in the associations between NO_2 and acute myocardial infarction is thoroughly discussed hereafter. Recent epidemiological studies have consistently shown the existence of relationship between NO_2 and increased hospital admissions due to heart disease. In a study by (Vencloviene et al., 2011), the authors investigated whether geomagnetic activity has an effect on the association between short-term exposure to NO_2 and emergency hospitalization for acute coronary syndrome (ACS). Vencloviene and colleagues observed that for patients under 65 years of age, an interquartile range increase (IQR) in ambient NO_2 pollution on the day of admissions and previous day (lag 0-1 day) increases the risk of acute coronary syndrome by 24%(95%CI:0.96-1.6). From these findings, the authors concluded that variations in geomagnetic activity may increases the effect of traffic related air pollution on acute coronary syndrome. In another study by (Stieb et al., 2009), the authors examined the associations between common air pollutants and respiratory or cardiac conditions for cities in Canada. Stieb and colleagues observed that an increase in 18.4ppb NO_2 was associated with a 2.6% increased hospital visits due to myocardial infarction/angina. From these findings, the authors concluded that daily mean levels of CO and NO_2 was most consistent with emergency department visits for cardiac conditions. In another study by (Stieb et al., 2009), the authors examined the associations between common air pollutants and respiratory or cardiac conditions for cities in Canada. Stieb and colleagues observed that an increase in 18.4ppb NO_2 was associated with a 2.6% increase in hospital visits due to myocardial infarction/angina. From these findings, the authors concluded that daily mean levels of NO_2 was most reliable for emergency department visits due to cardiac conditions.

Both these studies examined the daily concentrations of pollutants and hospitalizations due to myocardial infarction and both studies concluded that increased daily mean concentrations of pollutants is associated with increases in emergency hospitalization as well as increases in emergency department visits.

5.3.5 Mean concentrations of Sulphur dioxide

The sulphur dioxide levels in the Lautoka CBD was also well below the guidelines established by WHO (refer to Table 2). However, despite these low concentrations, epidemiological studies have shown that short-term exposure to SO_2 has an impact on the cardiovascular health. This was observed in the study by (Liu et al., 2017) where the authors conducted a time-stratified case crossover study in 14 large Chinese cities to examine the association of short term exposure to air pollution and daily admissions due to acute myocardial infarction. In this study Liu and colleagues observed that an interquartile range (IQR) increase in sulphur dioxide on lag - 2 days was significantly associated with a 2% increase in acute myocardial infarction hospital admissions. The authors of this study concluded that people who live in these 14 cities are at an increased risk of acute myocardial infarction hospitalizations if they are being exposed to SO2 even for a short-term. Moreover, Liu and others even suggested that the findings from this study can be applicable to developing countries as well. In another study by (Cheng, Tsai, & Yang, 2009), the authors investigated air pollutant levels increased the hospital admissions due to myocardial infarction in Kaohsiung, Taiwan. Their study yielded similar findings to that made by Liu and colleagues. Cheng and colleagues observed that in single pollutant models and on cool days (<25^{o}C) concentrations of SO_2 were positively associated with increased daily hospitalizations due to myocardial infarction. The authors of this study concluded that increased concentrations of pollutants such as SO_2 increases the risk of higher frequency of hospital admissions due to myocardial infarction. These studies suggest that the presence of SO_2-in the atmosphere can increase the risk of cardiovascular events leading to increased hospital admissions.

5.3.6 Weather variables (Temperature, Relative humidity and Atmospheric Pressure)

The weather data gathered during this study is typical of the day to day weather for locations on the western side of Viti Levu in Fiji. Since Lautoka CBD lies adjacent to the sea (refer to Figure 1), influences to the weather is mostly due to the surrounding ocean. In essence, Fiji weather is best described as tropical. This study coincided with the cooler season (May to November) persisting in Fiji with an average daytime temperature of just over 27oC. Environmental triggers of heart diseases have been under-recognized but have gained significant interest in the past decade (Pope et al., 1995). Of the three weather variables measured, ambient temperature has been consistently discussed in epidemiological studies to be associated with heart related diseases. For instance, in a study by (Claeys et al., 2015), the authors assessed the independent environmental triggers (air pollutants and weather variables) of acute myocardial infarction and to identify which sub-population are more vulnerable to these triggers. Claeys and colleagues observed that for temperature was significantly correlated with STEMI in that for every 10oC decrease in temperature an 8% increase in the risk of STEMI was apparent (adjusted incidence risk ratio (IRR) 0.92(95%CI: 0.89-0.96). From these findings, Claeys and others suggest that amongst the environmental triggers of STEMI, low temperature was the most important. Similar findings was observed in 2 other epidemiological study by (Royé et.al. 2018) and (Hori et.al. 2012). In the study by Roye and colleagues, they examined the relationship between ambient temperature and acute myocardial infarction hospital admissions in Cantabria, Spain. They observed an inverse but significant relationship between low ambient temperature and the number of hospital admissions due to acute myocardial infarction. From these findings, Roye and others suggest that environmental factor such as ambient temperature makes an important contribution to mortality due to acute myocardial infarction. In addition to examining the effects of air pollutants, Hori and colleagues also employed a time series study to explore the effects of weather variables namely, air pressure and ambient temperature on the emergency admissions

due to stroke and cardiovascular disease. They observed that for every 1^{o}C increase in ambient temperature there was a 7.83% increase in the daily admissions for acute coronary syndrome and heart failure, 95% CI: 2.06-13.25. They also observed that every 1hPa decrease in air pressure was associated with a 3.56%increase in emergency admissions due to heart failure, 95% CI: 1.09-5.96). All of the findings in the studies discussed are consistent in that ambient temperature plays an important role in the occurrence or triggering of cardiovascular events.

5.4 Overall and subgroup analysis of the systematic review and meta-analysis of studies

Recent epidemiological studies have reported the relationship between exposure to ambient air pollution and risk of hospitalizations due to acute myocardial infarction. Of the common air pollutants ambient particulate matter has gained wide concern, especially $PM_{2.5}$ (Luo et al., 2015). In this systematic review and meta-analysis, 20 case-crossover studies was identified that strictly met the inclusion criteria from 2008 to 2018. The included studies provided epidemiological evidence from 137,846 participants overall. Included studies (k = 20) exhibited effect estimates that had a positive association between exposure to ambient air pollution and risk of acute myocardial infarction hospital admissions or emergency department visits as well as the triggering of ST-elevated myocardial infarction. Since all studies in this meta-analysis do not share a common effect size and all effect sizes were sampled from a distribution of effects sizes, it is prudent to use the random effects model to report the estimated summary effect (Borenstein et al., 2009). Additionally and importantly, one of the objectives of this study is to extrapolate the outcome of this meta-analysis to a variety of scenarios such as the risk of AMI hospitalization for commuters to the Lautoka CBD due to exposure to air pollutants. The random effects model effect estimate was, OR = 1.0741(95%CI: 1.0490-1.0998). The overall analysis of these studies showed a high degree of heterogeneity, I^2 = 77%; Q statistics, 84.20(df=19; p<0.0001). Although this meta-analysis observed a high degree of heterogeneity, a meta-analysis study by (Cai, Li, Scott, Li, & Tang, 2016) suggest that such heterogeneity could be attributed to location of study and lag time used. The location of the selected studies and their different lag times are provided in Table 6 which shows varying lag times and locations. From the studies included in the meta-analysis, most number (5) of studies were from the United States of America, followed by Taiwan with 4 studies, 2 studies were from China and Sweden, and a study each from Japan, Italy, Canada, Belgium, France and Germany. In addition, other possible reasons for the high degree of heterogeneity as discussed by Cai and colleagues could be attributed to the selection of only studies with significant associations as well as differences in average particulate matter. Findings consistent with this study was observed in the two systematic review and meta-analysis studies by (Mustafić et al., 2012) and (Luo et al., 2015). The two studies examined the pooled estimates on the relationship between short-term exposure to common air pollutants and the risk of myocardial infarction based on a random-effects meta-analysis model. A moderate degree of heterogeneity was observed and also evidence of publication bias in $PM_{2.5}$ exposure. Authors from both these studies suggested that the risk of AMI was significant after short-term exposure to common air pollutants.

When compared with the overall pooled estimates, the results obtained from the two subgroup analyses were basically similar in that there was still significant association between exposure to air pollutants and risk of AMI hospitalization. However, when quantifying the level of heterogeneity, the degree of heterogeneity decreased from high in the overall analyses to low heterogeneity in both the subgroup analyses.

In the subgroup analysis on the risk of AMI hospitalization with short-term exposure to at least 5-10μg/m^3 increments of $PM_{2.5}$ was statistically significant; based on the random-effects meta-analysis model, a combined Odds Ratio (OR) = 1.0458(95%CI: 1.0267-1.0654), p<0.0001 was observed. A moderate degree of heterogeneity was detected, I^2 = 43% with evidence of publication bias. Similarly, the subgroup analysis on short-term exposure to common air pollutants and risk of AMI hospitalizations in Western Pacific based on the random-effects analysis model was also statistically significant with combined Odds Ratio (OR) = 1.05(95%CI: 1.03-1.08), p<0.0001. A low degree of heterogeneity was also observed, I^2 = 39%.

Furthermore, findings from the studies by (Weichenthal et al., 2017; Argacha et al., 2016; Chang, Kuo, Liou, & Yang, 2013; Zhang et al., 2016; Sahlén et al., 2019; Rich et al., 2013; Evans et al., 2016; Gardner et al., 2014; Hopke et al., 2015; Pope et al., 2015) are consistent with the findings from the subgroup analysis in that short-term exposure to 5-10μg/m^3 increments of particulate matter with an aerodynamic diameter of <2.5 micron ($PM_{2.5}$) is associated with an increased risk of hospital admissions due to acute myocardial infarction. The collective argument from these studies suggest that even at low concentrations, an increase of 5-10μ of $PM_{2.5}$ can trigger acute cardiovascular events like acute myocardial infarction and subsequently increase AMI hospitalizations.

In summary, based on the systematic review and meta-analysis of the 20 case-crossover studies, the pooled effect estimates in the overall analysis indicated a small but significant increased risk of AMI hospital admissions. In addition the subgroup the analyses suggests that exposure to at least 5-10μg/m^3 increments of $PM_{2.5}$ was significantly associated with an increased risk of AMI hospitalization.

5.5 Risk of increased AMI hospitalizations in Lautoka due to the ambient air quality in the Lautoka CBD

After carefully examining the results of the air quality data and the outcome of the meta-analysis, significant extrapolations can be made as to the risk of AMI hospitalisations in Lautoka. Of all the common air pollutants (PM_{10}, $PM_{2.5}$, O_3, SO_2, NO_2, CO) measured in the Lautoka CBD, only PM_{10} and $PM_{2.5}$ exhibited concentrations that was significant and relatable to the objectives of this study. Although the mean daily ambient $PM_{2.5}$ in the Lautoka CBD was relatively low (9.34µg/m^3) as shown in Figure 19, the outcome of the meta-analysis as well as that observed by (Mustafić et al., 2012) have shown that short-term exposure to low concentrations of the pollutant $PM_{2.5}$ is increases hospital admissions due to acute myocardial infarction. Consistent findings was also observed in the study by (Weichenthal et al., 2017), where it was found that short-term exposure to every 5µg/m^3 increments of a 3-day mean $PM_{2.5}$ was associated with an increased risk of myocardial infarction among the elderly.

Moreover, the daily PM_{10} mean concentrations exceeded the WHO recommended guideline value and poses a significant risk to the populace of Lautoka. Studies that shown the ill-effects of PM_{10} on the cardiovascular health include a case-crossover study by (Hsieh et al., 2010). In this study Hsieh and colleagues observed that after a 2 day lag, among other common air pollutants, PM_{10} was found to be statistically significant with increased hospital admissions due to myocardial infarction. Similar observations was made in the studies by (Liu et al., 2017) and (Yu et al., 2018)

The evidence gained in this study is that the ambient air quality in the Lautoka Central Business District is significant in particular both PM_{10} & $PM_{2.5}$ as described in numerous

epidemiological studies. The concentrations of particulate matter during this study with the evidence from recently published epidemiological studies suggests that the population of Lautoka are continuously exposed to high particulate pollution, especially PM_{10}.

Hence, from the evidence gained from epidemiological studies, it can be deduced that based on the overall analysis exposure to common air pollutants may increase the risk of AMI hospitalizations. In addition, every 5-10$\mu g/m^3$ increment in hourly or daily mean $PM_{2.5}$ concentrations can lead to increased hospital admissions due to acute myocardial infarction for commuters to the Lautoka CBD. Importantky, exposure to high PM_{10} concentrations and low $PM_{2.5}$ concentrations during peak traffic hours on Thursdays and Fridays may increase the risk of AMI hospitalization among the daily commuters to Lautoka city and its wider populace.

5.6 Limitations

A few setbacks was identified in terms of data collection and the analytical methods.

5.6.1 Air quality monitoring equipment.

The initial plan was to at least use 3 air quality monitoring sensors (AQMS) from the Geography Department (University of Canterbury) and be placed around the Lautoka CBD. However, on the final day of preparation, one of the sensors was not functional; hence only two AQMSs was used. During the study period, 1 of the 2 remaining AQMSs was not functioning as expected and I had to rely on moving 1 AQMS from 1 site to the next for 9 weeks. After 3 weeks into the data collection, the solar panels and the battery developed problems and had to be replaced with new ones bought in Fiji. Despite this drawbacks, sufficient air quality data was collected to satisfy one of the objectives of this study.

5.6.2 Health data

As discussed in the methodological design for this study, the short duration (3 months) of this study was assessed as a risk of not getting sufficient data. Sufficient health data would have enabled the adopting of an epidemiological design such as a case crossover design for this study and would have provided a more precise extrapolation. However, similar designed studies have also been used with significant contributions to the body of knowledge examining the association between exposure to air pollutants and risk of increased hospital admissions due to cardiovascular events.

5.6.3 Meta-analysis

The meta-analysis did not consider any other study design other than the case-crossover study design. The idea was to adopt a study design that focuses on short-term transient effects to air pollutants and this study design was sufficient. In addition, this thesis did not capture all studies that would have been captured for a meta-analysis and hence this thesis is biased towards studies that have only reported estimates on the basis of large sample sizes. The inclusion of other study designs into the meta-analysis would have yielded studies with small sample sizes and would have reduced the publication bias observed.

5.6.4 Time

Time was another limiting factor. This Master's degree by research allows a maximum of 3 months to collect data. Ideally more time would have led to more preparation time to identify all available and functional resources. Benefits of a longer period includes obtaining

sufficient data to increase the statistical power of the research. However, this would also meant an increase in funding allocation and for this study the budget was limited. Despite the time limits, I am quite satisfied with the outcome of this research and lessons learnt will definitely be adopted for continuation of this study in the future.

5.6.5 Modelling

This study did not employ complex statistical modelling because of the gaps in the data collected as a result of the malfunctioning of 2 of the air quality transmitters during the study.

CHAPTER 6

CONCLUSION

6.1 Re-examining of thesis objectives

This research has sufficiently addressed the main objectives of this Master's Thesis which are to:

1. Collect data on the ambient air quality in the Lautoka Central Business District for a period of three months

2. Determine the ambient air quality in the Lautoka CBD and its potential health implications

3. Conduct a meta-analysis of published studies on the association between ambient air quality and risk of hospitalization due to myocardial infarction.

4. Lastly, extrapolations on the risk of AMI hospitalizations on the basis of the meta-analysis and air quality data collected.

The key findings in this study includes the determination of the ambient air quality in the Lautoka CBD. As observed, the mean concentrations of PM_{10} in both the duration of the study as well as the daily means exceeded the guideline values set out by the World Health Organization. Although the mean concentrations of $PM_{2.5}$ was relatively low, such concentrations have been observed to increase AMI hospitalizations the world over. This study coincided with the sugarcane crushing season in Lautoka, hence, the presence of high concentrations of PM_{10} is indicative of high coarse materials emitted from the activities associated with the sugar industry. In addition, other contributing factors may include windblown dust and pollens from sugarcane plants.

$PM_{2.5}$ concentrations is indicative of the relative contributions mostly from combustion sources including vehicle emissions, open burning of waste and biomass burning. These emissions are mostly from vehicle emissions within the Lautoka CBD.

Both pollutants were significant in elevating the risk of AMI hospitalizations but it is the fine particulate matter, $PM_{2.5}$ based on epidemiological studies that poses the most significant risk to the triggering of AMI as well as the increased risk of hospital admissions.

6.2 Implications of results on the residents of Lautoka city

This thesis has provided a significant knowledge on the ambient air quality in the Lautoka CBD. More importantly is the evidence based knowledge that ambient air quality observed in this study can pose significant risks to the cardiovascular health of the wider populace of Lautoka city who are obviously the daily commuters. The results of this study will allow the vulnerable groups (children, elderly and patients with existing cardiac conditions) in the Lautoka populace to make informed choices on when is the best day or time of the day to visit the Lautoka CBD.

Moreover, to my knowledge this is the first epidemiological study to evaluate the impact of air pollutants on cardiovascular health in Fiji even without the availability of health data. However, the fact remains from reputable organization like the World Health Organization that exposure to air pollutant, specifically $PM_{2.5}$ increases the risk of cardiovascular events like acute myocardial infarction.

Furthermore, results of this study can provide the impetus for government organizations in Fiji to seriously consider ambient air quality as a significant health risk. Essentially, ambient air pollution has to be viewed as a potential risk factor to the cardiovascular system in addition to the traditional risk factors such as high blood pressure, high cholesterol level, unhealthy diet, physical inactivity and excessive consumption of alcohol. This is because heart disease is the major cause of premature deaths in Fiji and all risk factors has to be clearly identified and addressed in its entirety to minimize loss of lives.

The findings of this study therefore contribute to the limited literature available on ambient air quality and risk of acute myocardial infarction hospitalizations in Fiji

6.3 Future directions

This study has provided potential opportunities that warrants further investigation into the ambient air quality in Fiji where sugarcane activities are rife. This is basically for all towns in the Western division including Labasa from the Northern division of Fiji. Understanding the ambient air quality in these locations will provide sufficient information to determine the levels of risks as well as identifying the susceptible groups.

Further studies to validate the findings from this research must be conducted but in a longer time scale and with inclusion of health data. These studies will not only be for academic purposes but to aid in developing policies that will curtail emissions from industries, vehicles as well from other anthropogenic means.

Finally, Fiji does not monitor ambient air quality resulting from potentially economic reasons or from lack of international support. More epidemiological studies like this research can provide the momentum of recognizing the importance to monitor ambient air quality at least in urban centers.

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APPENDIX 1: DATABASE SEARCH STRATEGY

Database: Ovid MEDLINE(R) and In-Process & Other Non-Indexed Citations and Daily <1946 to November 27, 2018> Search Strategy:

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1 air pollution/ or air pollutants/ (60303)
2 particulate matter/ (13893)
3 Vehicle Emissions/ or Ozone/ or Carbon Monoxide/ (38292)
4 Sulfur dioxide/ (5012)
5 nitrogen dioxide/ (4329)
6 (air pollut* or air quality or particulate matter or vehicle emission* or sulphur dioxide or sulfur dioxide or "PM2.5" or "PM10").ti,ab,kf. (46838)
7 1 or 2 or 3 or 4 or 6 (110933)
8 exp Myocardial Infarction/ (166138)
9 Acute Coronary Syndrome/ (13184)
10 (myocardial infarction or heart attack or heart infarction).ti. (71772)
11 8 or 9 or 10 (182243)
12 7 and 11 (374)
13 (letter or editorial).pt. (1472972)
14 12 not 13 (347)
15 limit 14 to (english language and yr="2008 -Current") (205)

Database: Embase <1974 to 2018 December 10>
Search Strategy:

---

1 air pollutant/ (29445)
2 air pollution/ or nitrous oxide emission/ (55418)
3 carbon monoxide/ (33407)
4 ozone/ (25494)
5 exhaust gas/ (17679)
6 sulfur dioxide/ (13638)
7 particulate matter/ (36720)
8 (air pollut* or air quality or particulate matter or vehicle emission* or sulphur dioxide or sulfur dioxide or "PM2.5" or "PM10").ti,ab. (65479)
9 or/1-8 (173203)
10 acute heart infarction/ or myocardial infarction/ or heart infarction/ or acute coronary syndrome/ (342892)
11 (myocardial infarction or heart attack or heart infarction).ti. (98735)
12 10 or 11 (354367)
13 9 and 12 (1191)
14 limit 13 to (english language and yr="2008 -Current") (871)
15 animal/ not human/ (1017266)
16 14 not 15 (867)
17 limit 16 to (books or chapter or editorial or letter or note) (125)
18 16 not 17 (742)

APPENDIX 2: THESIS APPROVAL LETTER

UNIVERSITY OF CANTERBURY
Te Whare Wānanga o Waitaha
CHRISTCHURCH NEW ZEALAND

HUMAN ETHICS COMMITTEE
Secretary, Rebecca Robinson
Telephone: +64 03 369 4588, Extn 94588
Email: human-ethics@canterbury.ac.nz

Ref: HEC 2018/44/LR

23 July 2018

Josefa Tabua
School of Health Sciences
UNIVERSITY OF CANTERBURY

Dear Josefa

Thank you for submitting your low risk application to the Human Ethics Committee for the research proposal titled "Ambient Air Quality and the Risk of Acute Myocardial Infarction Among Urban Dwellers in Fiji".

I am pleased to advise that this application has been reviewed and approved.

Please note that this approval is subject to the incorporation of the amendments you have provided in your email of 16th July 2018.

With best wishes for your project.

Yours sincerely

pp. R. Robinson

Professor Jane Maidment
Chair, Human Ethics Committee

APPENDIX 3: R script for data pre-processing

```
library(tidyverse)
library(readxl)
library(lubridate)

aqdata <- read_excel("air_quality_data/air_quality_data/Master_Sheet.xlsx")
str(aqdata)
View(aqdata)


aqdata_time_corrected <- aqdata %>%
  mutate(hour = hour(Time),
         minute = minute(Time),
         second = second(Time),
         date = as_date(Date),
         year = year(date),
         month = month(date),
         day = day(date))

 aqdata_time_corrected %>%
   str()

 # group by the data based on hour and then based on day

 aqdata_revised <- aqdata_time_corrected %>%
   group_by(month, day, hour) %>%
   summarise(mean_temp = mean(Temp., na.rm = T),
             mean_humidity = mean(Humidity, na.rm = T),
             mean_pressure = mean(Pressure, na.rm = T),
             mean_no2 = mean(NO2, na.rm = T),
             mean_so2 = mean(SO2, na.rm = T),
             mean_co = mean(CO, na.rm = T),
             mean_ozone = mean(O3, na.rm = T),
             mean_pm25 = mean(PM2_5, na.rm = T),
             mean_pm10 = mean(PM10, na.rm = T))

 # aqdata_revised$datenum <- as.numeric(aqdata_revised$date)

 View(aqdata_revised)
```

APPENDIX 4: R script for Meta-analysis

```
library(tidyverse)
library(meta)
```
```{r}
mydata <- read.csv("Odds_Ratios_adjusted.csv")
```
```{r}
view(mydata)
```
```{r}
mydata2 <- mydata %>%
  select(Author,OR,N,quality_score,logOR,se_OR)
```
```{r}
View(mydata2)
```
```{r}
mydata2$se_OR = as.numeric(mydata2$se_OR)


```
```{r}
my_meta = metagen(logOR,se_OR,
                  data = mydata2,
                  sm = "OR",
                  studlab = Author
                  )
```
```{r}
summary(my_meta)

```
```{r}
forest(my_meta)
```
```{r}
funnel(my_meta)
```